Surgery of the Foot

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Foreword by

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Introduction by

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WITH 403 FIGURES

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TO

Frances K DuVries, My Wife

Foreword

FOR MANY YEARS I HAVE BEEN CONCERNED WITH THE LACK OF TEACHING OF medical students and physicians in the care of the feet I have witnessed this paucity of teaching in the surgical curricula of the university medical schools and in the teaching programs of the residents in surgery and orthopedics in large municipal hospitals

Few students and practitioners seem interested in this important subject It is a tribute to the physician and surgeon whose consuming interest is with this particular problem The modern student is far more interested in the rare surgical conditions as exemplified by cardiovascular surgery and the radical procedures in the treatment of cancer Yet, a great deal of his early practice will be concerned with diseases of the extremities and especially of the feet

I salute Dr Henri DuVries, who has made the foot his life's study and who is now presenting many of his observations and practical experience in this particular specialty The profession can profit by observing his methods and the results of his treatment of the common disorders of the foot. He early recognized that simplicity does not diminish distress, and so he set out to enlist interest in this neglected phase of medicine and surgery He did this by precept and example. he limited his practice to the correction of disabilities of the foot, and he concentrated his teaching on that subject After a close association of many years, and as a colleague, whom I sponsored at the time of his admission to the International College of Surgeons, I highly endorse his teaching, for I have personal knowledge of his successful application

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Introduction

THE NUMBER OF SURGEONS WHO DEVOTE MOST OR ALL OF THEIR PROFESSIONAL time to diagnosis and treatment of disabilities of the human *hand* has been increasing during the last twenty years The specialist in surgery of the hand is highly regarded, and those who are primarily interested in the diseases, injuries, and deformities of the hand have formed a National Society for Hand Surgery In contrast, although several books describing the anatomy, physiology, injuries to, diseases of, or deformities of the foot have been published, the authors of these books were not necessarily specialists who limited their interests to the *foot* and the diseases or injuries which may disturb its function One outstanding exception to this situation is Dr Henri L DuVries who for nearly thirty years has devoted his time and energies to a better understanding of the structure and function of the foot and to alleviation of the disabilities which may affect it

Here then is a reference book which should prove helpful to orthopedic surgeons, general surgeons, pediatricians, and physicians in general practice who are called upon to treat an abnormal condition of a human foot Over 400 illustrations, including line drawings, shaded drawings, photographs, and roentgenograms, illuminate the text

The opening chapter of this book begins with a description of the structure and the function of the bones of the ankle and of the muscles of the calf In Chapter 2, Dr DuVries tells how to examine the human foot to make a diagnosis which will explain the cause of the patient's symptoms Included in the equipment which he considers essential for an adequate examination and accurate diagnosis, he lists *jewelers' eyeglasses* through which minute defects in the skin or in the contour of the foot may be more readily detected He also recommends that the skeleton of a normal foot be at hand for study in comparison with roentgenograms of the feet of each patient The general principles of operations upon the foot as described and the many common-sense suggestions, which are the outgrowth of the wide and diligent experience of the author, will be helpful to all who are confronted with similar problems

Preface

THIS BOOK HAS BEEN WRITTEN IN RESPONSE TO A CONTINUING REQUEST BY MY students and colleagues that I draw together into one place of reference the fundamentals and the recommendations contained in my lectures and clinical demonstrations over a span of thirty years As Frederic Wood Jones commented, "It is probably the experience of most teachers of anatomy that the student is generally better acquainted with the intimate structure of the hand than with that of the foot "[*] My friends among orthopedic surgeons agree that the teaching of their specialty does not allot sufficient time to problems of the foot They will forgive, therefore, and perhaps welcome as an adjunct to teaching, the elementary portions of the contents and the didactic approach

Extreme disabilities of the foot, such as the talipes deformities, have received studious attention in published reports They have on that account been given only a cursory nod of recognition here This book is directed toward the commoner disabilities, which have been sparsely considered in medical writing and which have been widely neglected in teaching and practice

The expanding awareness of the diversity of pathologic changes in the feet and of the complexities of treatment represents an advance since the days when all foot disabilities were always attributed to so-called fallen arches and when a prescription of arch supports satisfied the diagnostician that nothing further could be done about the patient whose feet continued to hurt

This far from definitive effort of mine has reached the printed page through the encouragement and helpfulness, advice, and direction of so many of my friends and colleagues that I hesitate to name them lest by inadvertence one should be overlooked If that happens, my deepest regret! Certainly I must mention my friends of long standing, Dr August F Daro, Dr William M Scholl, who turned his collection of photographs and anatomic models over to me for study and selective use and who has been otherwise helpful in so many ways, Dr

[*] Jones, F W Structure and Function as Seen in the Foot, London, 1944, Baillière, Tindall & Cox, Ltd , p 3

Ernest Nora, Sr , a constant friend since our medical schools days, who reviewed the chapter on Tumors, Cysts, and Exostoses, so many on the Staff of Columbus Hospital, Dr Carlo Scuderi, who reviewed the first rough material and then introduced me to my patient and cooperative publishers, Dr Edwin Hirsch, who made the photographic facilities of St Luke's Hospital[*] available to me, Dr Karl A Meyer, my former professor in medical school, who wrote the Foreword as a final expression of years of encouragement Dr Edward L Compere crowned my effort by writing the Introduction, having first reviewed some of the material in its early stages and, later, all in its final form

Special credit should be given to Miss Ethel H Davis for superbly editing and organizing the manuscript

It is tempting to list those who gave me direction in one way or another Dr Peter A Rosi, Dr Charles N Pease, Dr Joseph P Cascino, Dr Steven O Schwartz, Dr Caesar Portes, Dr Abe Rubin, and Dr Harold Wheeler The skill of my artists, Miss Edith Hodgson, Miss Gloria Jones, and Dr Allen Whitney, must not go unsung And to all, mentioned or not, in the measure of their interest, my gratitude!

Only wives whose husbands have attempted the writing of books and the husbands who have known the stamina of their wives during the process can appreciate how much meaning there is in my dedication to Frances DuVries

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Surgery of the Foot

CHAPTER ONE

Structure and Function

STRUCTURE

STRUCTURES EXTRINSIC TO THE FOOT WHICH INFLUENCE ITS FORM AND FUNCTION and which are revelant to surgical procedures for foot disorders are (1) the bones, particularly those of the ankle; (2) fascia and ligaments of the ankle, including the interosseous tibiofibular ligament, all of which are stabilizers of the foot and ankle, and (3) the muscles of the leg, which are essential to the kinetic functions of the foot For the sake of convenience in orientation, these structural relationships of the bones and fascia of the ankle are briefly reviewed The muscles of the leg are illustrated without discussion (Figs 1 to 7) Recapitulation is not intended to supplant detailed textbooks on anatomy.

Bones of the Ankle

The ankle is composed of the lower end of the tibia and its extended tip, the medial or internal malleolus, and the lower end of the fibula. The lower end of the fibula forms the outer or lateral malleolus The malleoli and the inferior transverse tibiofibular ligament hold the trochlear surface and sides of the body of the talus in a mortise, permitting a normal movement of about 60 degrees between dorsiflexion and plantar flexion Because of the locking mechanism of the lower end of the tibia and medial malleolus with the talus, little if any eversion takes place, however, there is latitude for some inversion as the lateral malleolus is not a fixed part of the mortise (Fig 8)

Fascia and Ligaments of the Ankle

Fibrous bands, or thickened portions of the fascia, envelop and bind down the tendons of the muscles as they pass from the leg in front of the ankle into

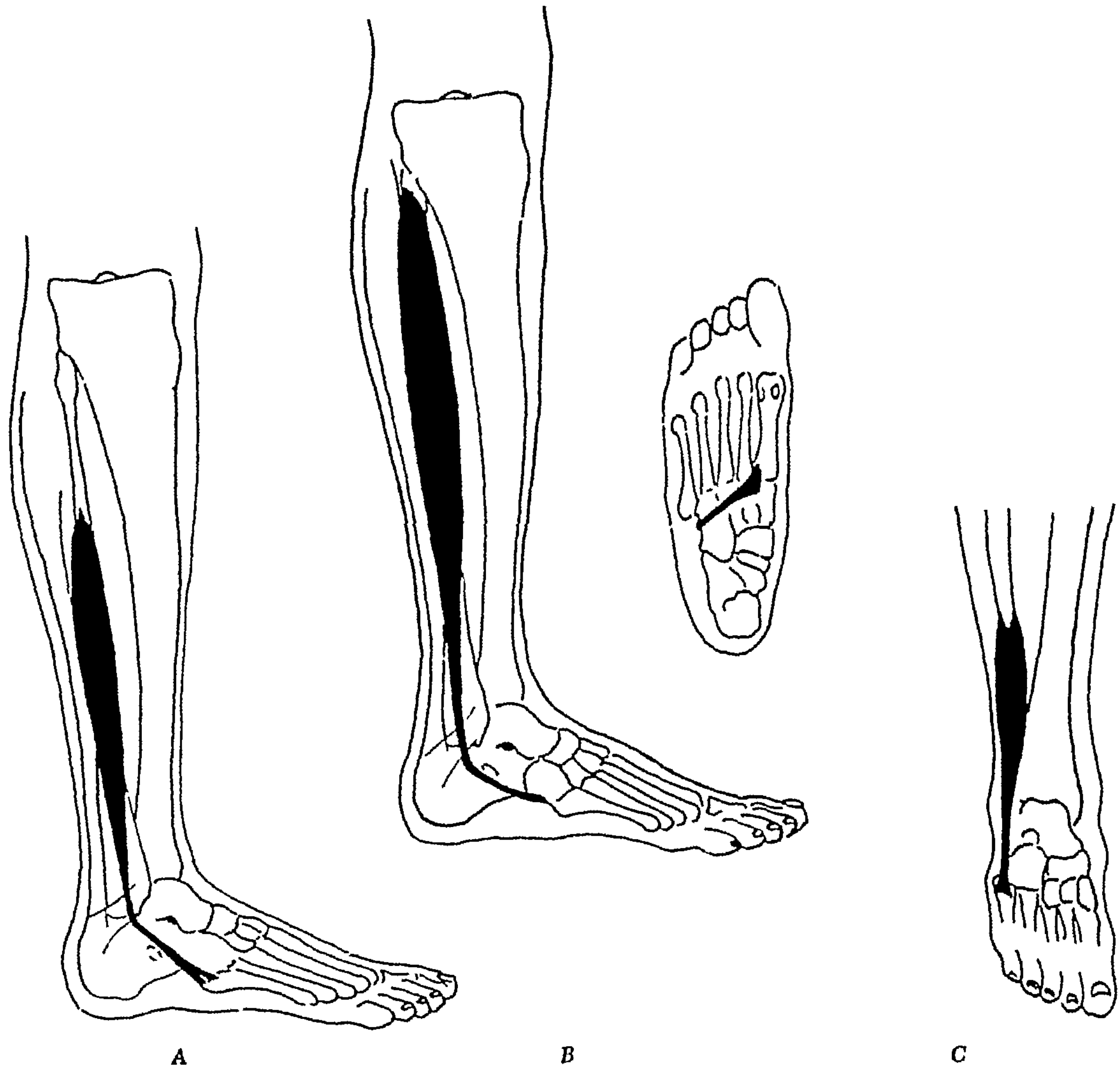


Fig 4—Peroneal muscles A, Brevis, which is inserted into base of fifth metatarsal abductor of foot B, Longus, small sketch showing insertion of longus muscle into plantar surface of first cuneiform and base of first metatarsal abductor and everter of foot C, Tertius, insertion into dorsal surface of base of fifth metatarsal dorsiflexor of foot

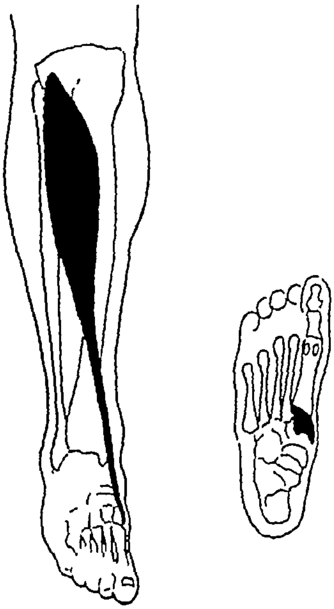


Fig 1 —Tibialis anterior and its insertion, act as inverter of foot

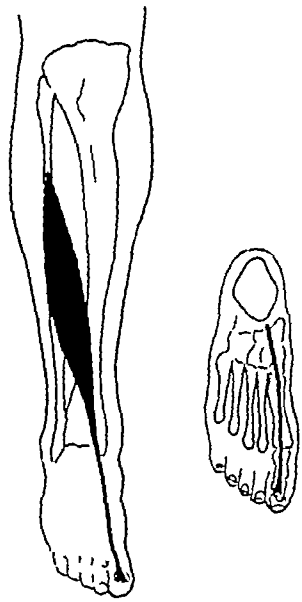


Fig 2 —Extensor hallucis longus and its insertion into distal phalanx and great toe, dorsiflexor of both toe and foot

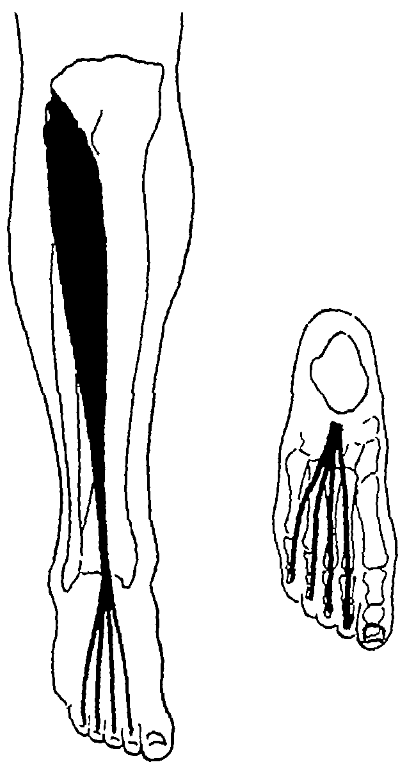


Fig 3 —Extensor digitorum longus with its insertion in four lesser toes, dorsiflexor of lesser toes as well as of foot.

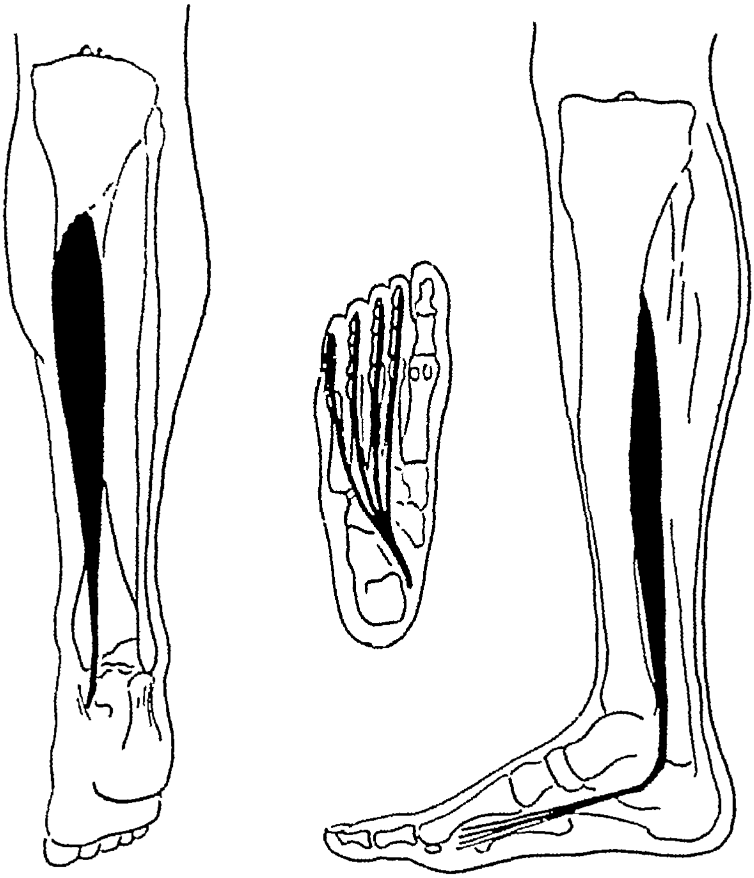


Fig 7.—Flexor longus digitorum with its insertion into plantar surface of four lesser digits plantar flexor of toes and foot

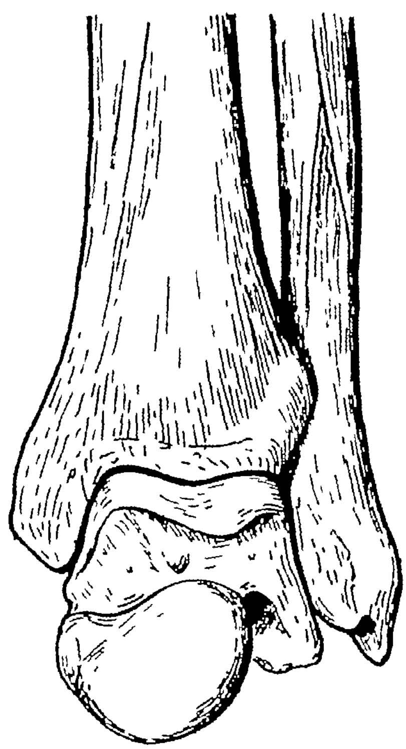


Fig 8 —Ankle mortise

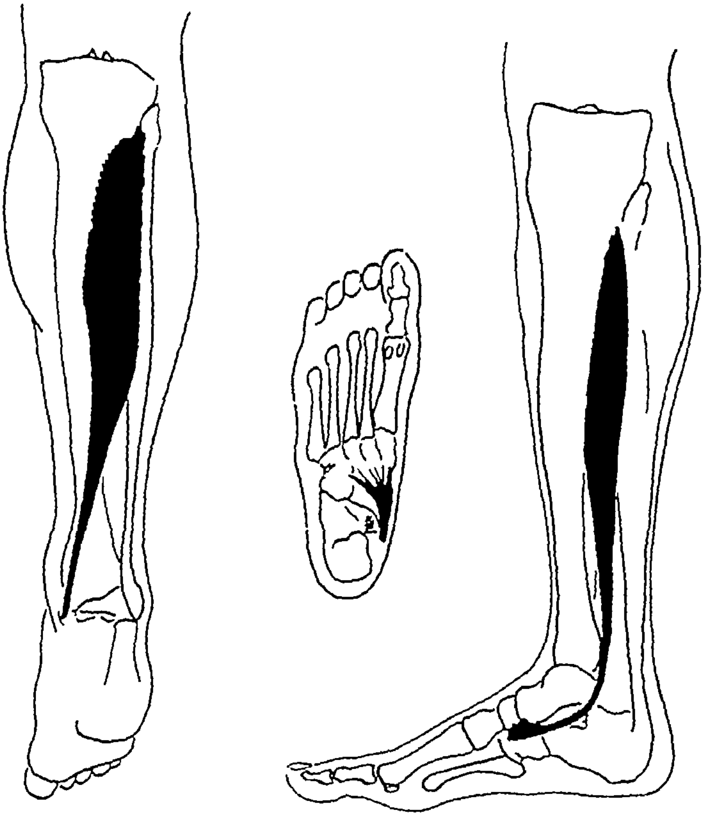


Fig 5 —Tibialis posterior with its insertion into plantar surface of navicular plantar flexor and adductor

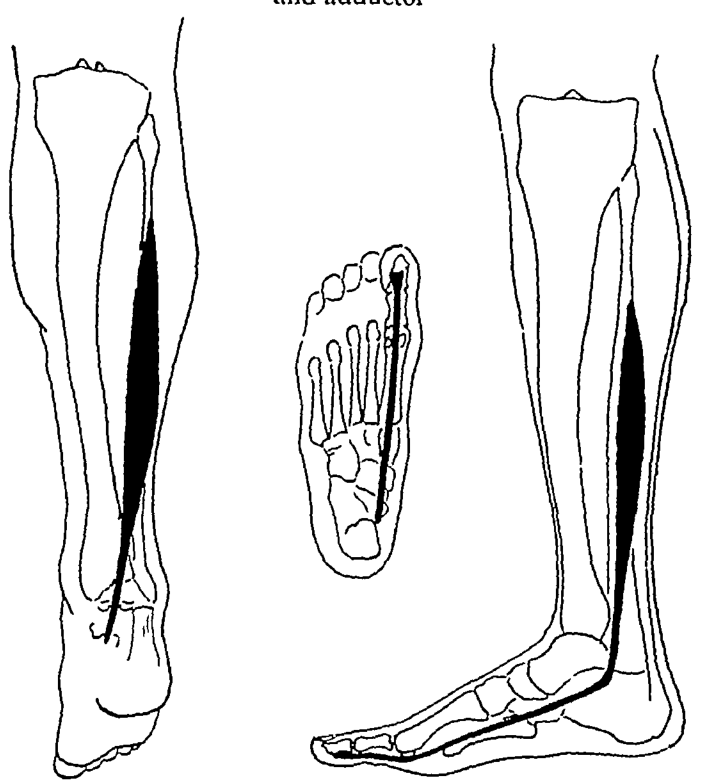
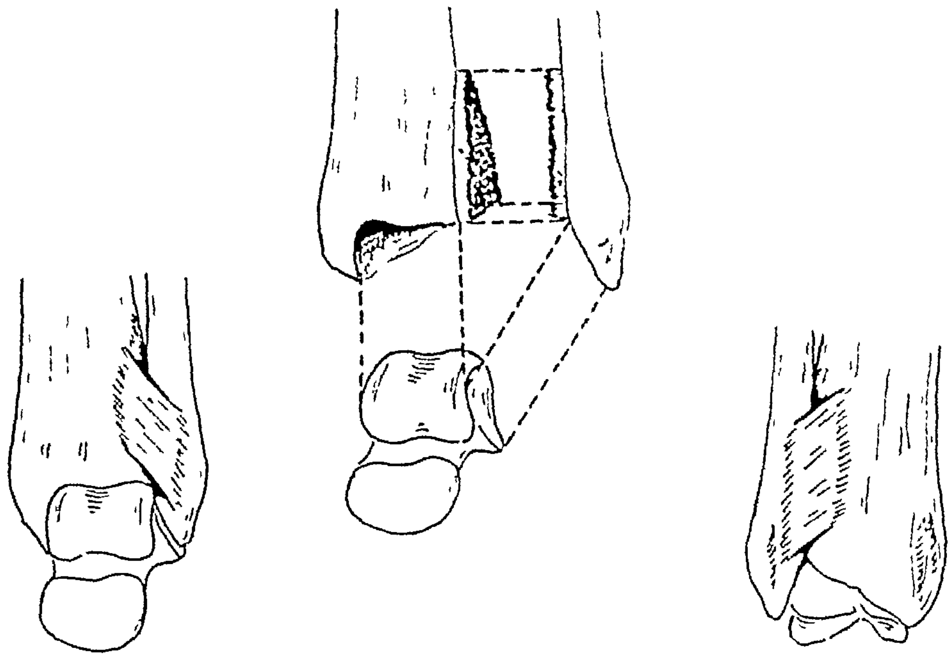


Fig 6 —Flexor hallucis longus with its insertion into plantar surface of distal phalanx of great toe plantar flexor of great toe and foot

Peroneal Retinacula.—The peroneal retinacula are two fibrous bands, the superior retinaculum and the inferior retinaculum, which reach across the lateral side of the ankle to bind down the tendons of the peroneus longus and peroneus brevis. The fibers of the superior retinaculum are attached to the lateral surface of the calcaneus.

Tibiofibular Articulation.—It is also pertinent to mention the inferior tibiofibular articulation, or the tibiofibular syndesmosis, which is held together (Fig 10) by the anterior tibiofibular ligament, the posterior tibiofibular ligament, and the interosseous ligament.



A *B* *C*

Fig 10—Tibiofibular ligaments Note disruption of ankle mortise when these ligaments are torn *A*, Posterior view. *B*, Rupture of the syndesmosis *C*, Anterior view

Bones of the Foot

The twenty-six bones of the foot are divided into three groups: the seven tarsals of the tarsus, the five metatarsals of the metatarsus, and the phalanges of the toes, of which the great toe has two and each of the others has three (Figs 11 to 14).

Tarsus.—The seven bones composing the tarsus, or instep, are in the posterior, or proximal, part of the foot, they are arranged in two rows with one bone between them In the posterior row are the talus and calcaneus, the intermediate bone is the navicular, in the anterior row are the cuboid and the first, second, and third cuneiforms

Talus—The talus, or astragalus, variously called the *ankle* or *knuckle* or *saddle* bone, rests on the calcaneus and is gripped by the medial and lateral malleoli The talus and malleoli form the *ankle mortise* The largest part of the

the dorsum of the foot The fascia is composed of four ligamentous structures· the transverse crural ligament, the cruciate crural ligament, the laciniate ligament, and the two peroneal retinacula (Fig. 9)

Transverse Crural Ligament.—The transverse crural ligament stretches across the lower end of the tibia and fibula to bind down the tendons of the extensor digitorum longus, extensor hallucis longus, peroneus tertius, and tibialis anterior It is attached laterally to the lower end of the fibula and medially to the tibia.

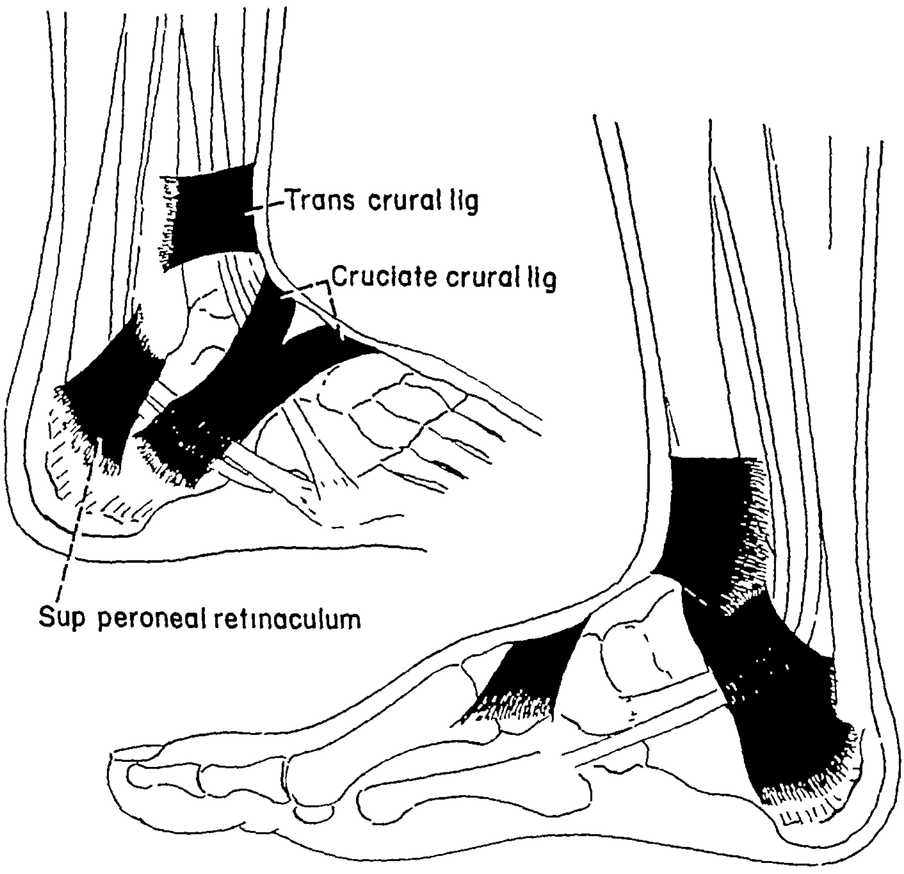


Fig 9 —Superficial ligaments of ankle binding tendons of leg as they course into foot

Cruciate Crural Ligament —The cruciate crural ligament is a Y-shaped band in front of the ankle joint, having the stem of the Y attached to the upper surface of the calcaneus Directed medially in two distinct layers, one portion passes in front of, and the other passes behind, the tendons of the peroneus tertius and extensor digitorum longus From the medial part, the two limbs of the Y fork out, one extending upward and medialward to attach to the tibial malleolus, and the other extending downward and medially and fusing with the medial part of the plantar aponeurosis

Laciniate Ligament.—The laciniate ligament, a strong, fibrous band, crosses the hollow between the medial malleolus and the medial prominence of the calcaneus and is attached to both It is continuous at its upper border with the deep fascia of the leg and at its lower border with the plantar fascia, binding the tendons of the tibialis posticus, flexor hallucis longus, and flexor digitorum longus

Peroneal Retinacula.—The peroneal retinacula are two fibrous bands, the superior retinaculum and the inferior retinaculum, which reach across the lateral side of the ankle to bind down the tendons of the peroneus longus and peroneus brevis The fibers of the superior retinaculum are attached to the lateral surface of the calcaneus.

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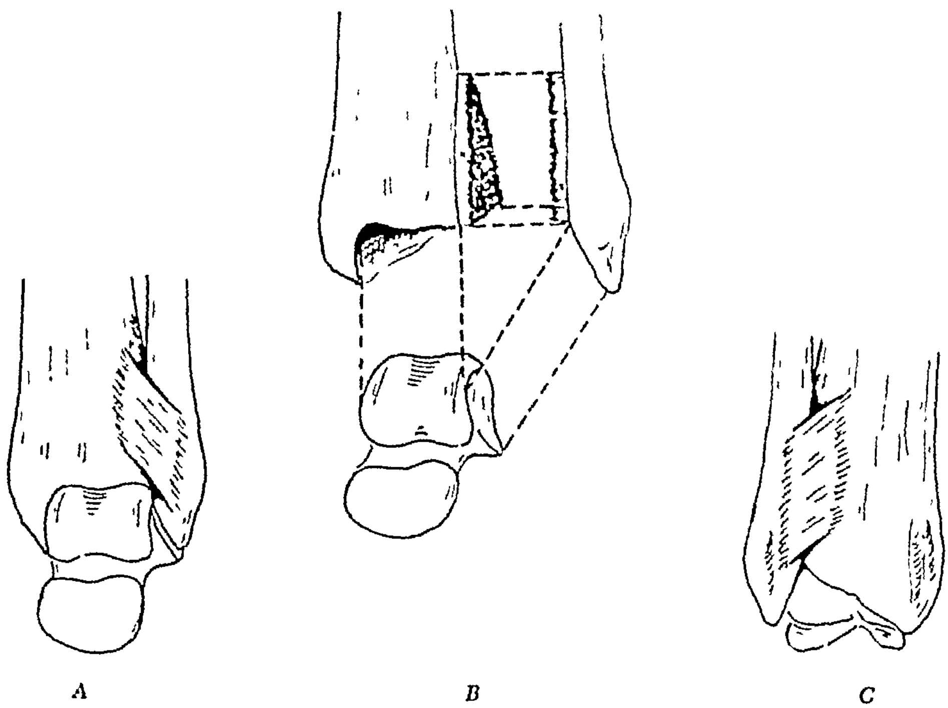

Fig. 10 —Tibiofibular ligaments Note disruption of ankle mortise when these ligaments are torn *A*, Posterior view *B*, Rupture of the syndesmosis *C*, Anterior view

Bones of the Foot

The twenty-six bones of the foot are divided into three groups: the seven tarsals of the tarsus, the five metatarsals of the metatarsus, and the phalanges of the toes, of which the great toe has two and each of the others has three (Figs 11 to 14).

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talus is the *body,* the smooth, rounded projection is the *head,* the area constricted in between is the *neck.* The anterior surface of the head is convex and smooth for articulation with the navicular. The posterior surface is small and has a diagonal groove across it in which rests the tendon of the flexor hallucis longus muscle. The superior surface forms a large convex facet, the *trochlea,*

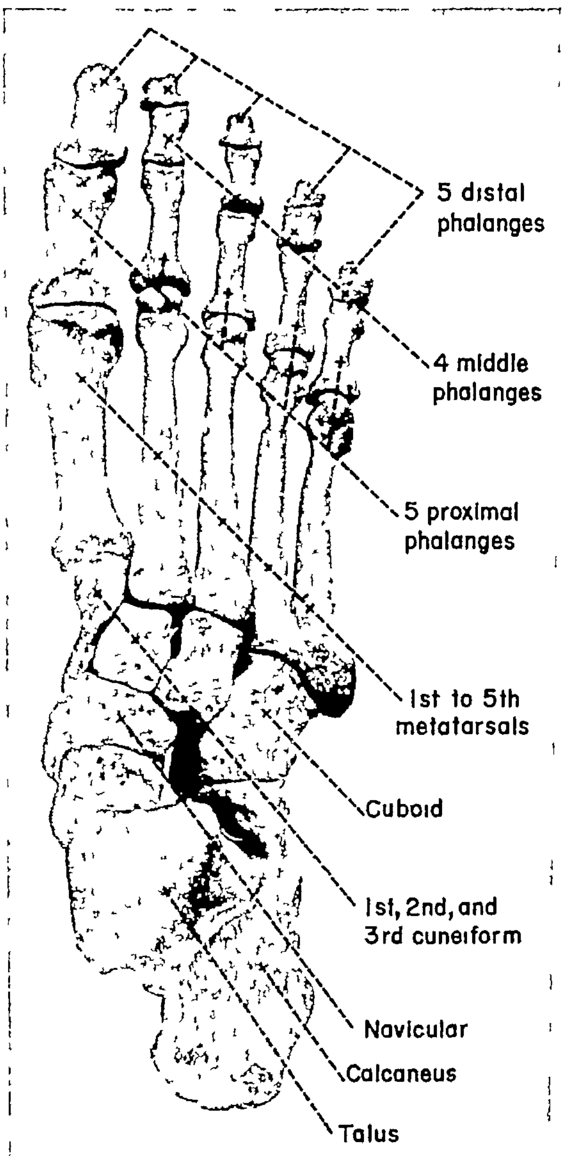

Fig. 11.—Bones of foot, dorsal view.

for articulation with the lower end of the tibia. A facet on each side of the superior surface articulates with the malleoli. The inferior surface has two facets, a deep groove between them is called the *sulcus tali.* The lateral facet of the inferior surface is the larger of the two and articulates with the superior facet on the calcaneus, the medial facet is small and articulates with the sustentaculum, the talus-supporting process of the calcaneus.

Only 25 per cent of the surface of the talus can receive blood because the rest of its surface articulates with other bones, its blood supply, therefore, is more easily impaired than that of any other bone in the foot.

Calcaneus—The calcaneus, or *os calcis*, the largest bone of the tarsals, measures about 3½ inches in length and about 1½ inches at its widest point. The an-

Fig 12—Bones of foot, plantar view

terior surface articulates with the cuboid The superior surface at the anterior part of the bone has a large, smooth facet for articulation with the body of the talus On the medial side of the calcaneus is a shelf of bone that projects medially, presenting a facet on its superior surface This is the *sustentaculum* already referred to in discussing the talus, and it likewise articulates with the talus Beneath the sustentaculum is a groove accommodating the tendon of the flexor hal-

lucis longus muscle The posteroinferior surface of the calcaneus is prominent and convex, forming the calcaneal tuberosity. Into its posterior aspect the tendo achillis and plantaris are inserted. Anterior to the inferior surface is a sulcus, from which the plantar fascia arises.

Navicular—The navicular, or *scaphoid*, surfaces are the anterior, which is smooth and slightly convex and divided into three areas of articulation with the three cuneiform bones, the posterior, which is smooth and concave and articulates with the head of the talus, and the medial surface, which forms a rough projection called the *tuberosity of the navicular*

Fig 13—Bones of foot, medial view

Fig 14—Bones of foot, lateral view

Cuboid—The cuboid is a keystone-shaped bone Its anterior surface articulates with the bases of the fourth and fifth metatarsals, its posterior surface articulates with the head of the calcaneus On the anteroinferior surface is a groove in which is lodged the tendon of the peroneus longus muscle It forms a keystone between the head of the calcaneus and the base of the fifth metatarsal

Cuneiform—The three cuneiform bones lie between the navicular and the first three of the metatarsal bones The medial cuneiform, or first internal bone, is the largest, it articulates on its anterior surface with the base of the first metatarsal bone The posterior surface articulates with the navicular The lateral surface has facets for articulation with the intermediate cuneiform and the base of the second metatarsal The intermediate, or second middle, cuneiform is the

smallest bone in the tarsus. Its anterior surface articulates with the second metatarsal, its posterior surface articulates with the navicular; its medial surface has a large facet for articulation with the lateral cuneiform. The lateral, or third external, cuneiform has a facet on its anterior surface for articulation with the navicular, and the medial surface articulates with the intermediate cuneiform The lateral surface has a small facet along its anterior border for articulation with the cuboid.

Metatarsus.—The five metatarsal bones are numbered one to five, beginning with the medial metatarsal. The metatarsal bones are miniature long bones having a shaft and two ends The distal end, or head, is rounded, the proximal end, or base, is flat At their bases the metatarsals are firmly bound to the tarsus. The lateral four articulate with one another at their bases. The first may articulate with the second but is ordinarily free. The first metatarsal bone is the thickest of the five It has two small facets on the plantar surface of the head for articulation of the two hallux sesamoid bones. (The sesamoids are discussed as accessory bones in the next section of this chapter. Diseases of the sesamoids are discussed in Chapter 12)

The second, third, and fourth metatarsals are similar The plantar surface of their heads has two sharp condylar projective points which are characteristically sharper on the fibular side The fifth metatarsal has a small head and a large base, it articulates with the cuboid, a large tubercle is on the lateral side of its base

Phalanges.—The two large phalanges of the great toe are designated *proximal* and *distal.* The extensor hallucis longus and flexor hallucis longus are inserted into the distal phalanx, and the extensor hallucis brevis and the abductor and adductor hallucis muscles are inserted into the base of the proximal phalanx. The other four toes each have three phalanges. proximal, middle, and distal The phalanges are shaped like miniature long bones, the proximal phalanx being the longest and the distal, the smallest

Accessory Bones of the Foot

Sesamoids.—The sesamoids were so named because of their resemblance to sesame seeds The sesamoids are not true accessory bones, they are part of the normal skeleton, and the normal foot has two of them They are discussed here rather than among the twenty-six bones of the foot because of their development in a tendon They are incorporated in the tendon of the flexor hallucis brevis, proximal to its insertion into the plantar surface of the base of the first proximal phalanx The tibial sesamoid is convex transversely and concave longitudinally The fibular sesamoid is usually flat transversely and concave longitudinally The sesamoids vary, however, in size and shape

The sesamoids articulate with the plantar surface of the head of the first metatarsal in sulci formed for them The tibial sesamoid is usually the weight-bearing pivot of the great toe joint, only occasionally does the fibular sesamoid touch the ground

Types of Accessory Bones.—When the human foot deviates from the normal, it is mostly in its accessory bones. Some accessory bones are additional sesamoids, small ones in tendons, some accessory bones are independent anomalies, varying in size, shape, and frequency of occurrence. Ordinarily, accessory bones are prominences of the bones of the foot, separated either abnormally from the main elements or as subdivisions of the main elements In some instances they have separate centers of ossification. In general, such skeletal variations elsewhere in the body are only of academic interest, in the foot, however, their interest is a practical one When accessory bones become detached or loosened from their original position or when their size or shape produces friction, disability results.

Fig 15 —Accessory navicular

In Shands' (1953) series of 850 roentgenograms of children having undiagnosed foot disabilities, accessory bones were disclosed in 115, in the following order of frequency 36 accessory navicular (Fig 15), 28 os vesalianum (Fig 16), 27 os trigonum (Fig 17), 7 os supranavicular* (Fig 18), 4 os intercuneiform, 3 os intermetatarsum (Fig 19), 3 os peroneal (Fig 20), 2 os subfibula, 2 os subtibiale, 1 talonavicular* ossicle (Fig 21), 1 secondary calcaneus, and 1 os subcalcis Most accessory ossicles of the foot are recognized and have been described by anatomists (Fig 22)

Watkins (1937) directed attention to the frequency with which accessory ossicles of the foot are mistaken for fractures Pirie (1921) first described the

*Refer to the same bone

Fig 16

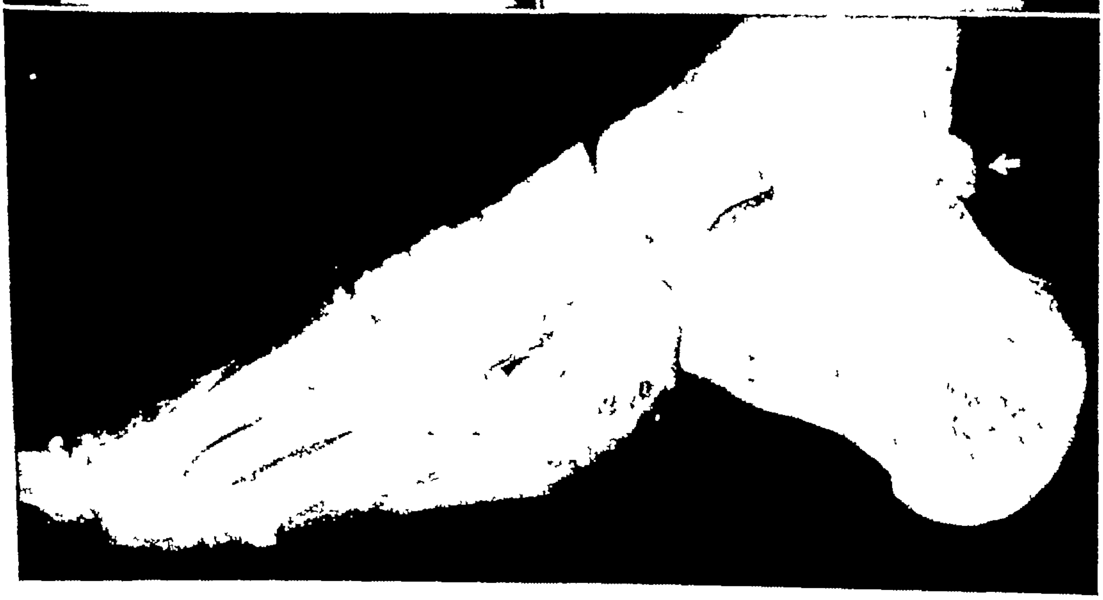

Fig 17

Fig 16 —Os vesalianum

Fig 17 —Os trigonum

Fig 18

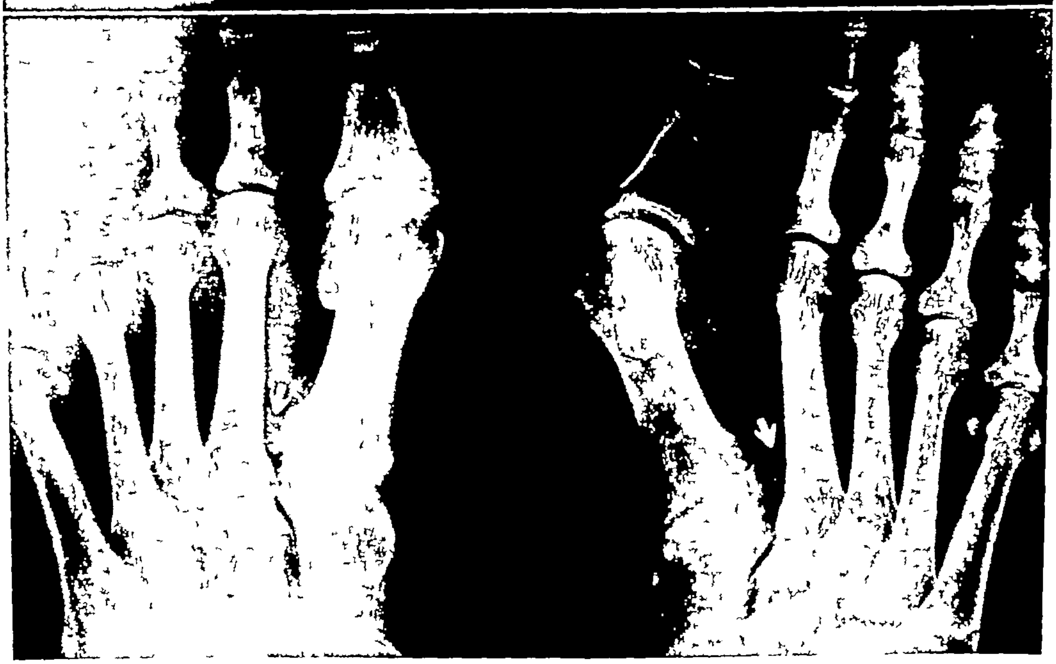

Fig 19

Fig 18 —Os supranavicular

Fig 19 —Os intermetatarsum

Fig 20

Fig 21

Fig 20 —Os peroneal

Fig 21 —Os talonavicular (Pirie's bone)

accessory bone (Pirie's bone) on the dorsum of the talonavicular (Fig 21), and he also observed that this ossicle is generally mistaken for fracture Years later Gottlieb and Beranbaum (1950) made the same observation

March and London (1956) searched for published reports and record that "there have been thus far 4 cases of os sustentaculi and 6 cases of (related) articulatio talo-calcanea . reported altogether" They cite Shanks and Kerley

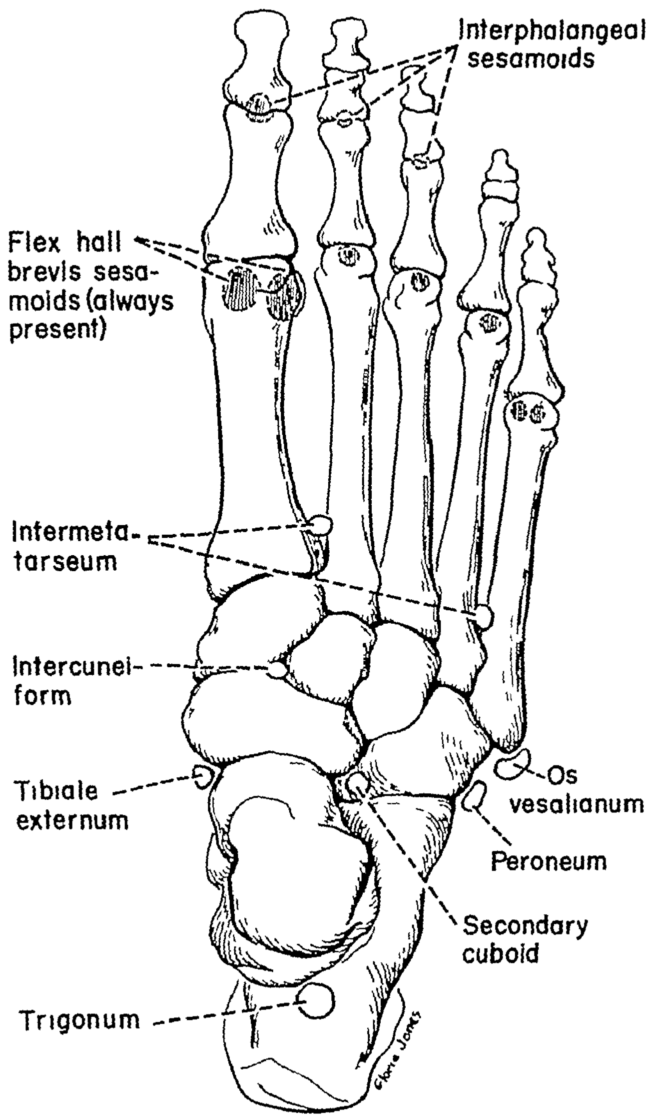

Fig 22 —Schematic drawing of accessory bones

as observers who regard "its existence as doubtful" March and London, however, encountered two cases within two weeks and added their reports They describe the ossicle as appearing "at the posterior end of the sustentaculum tali" and they report that it "produces a small visible and palpable projection just below and very slightly posterior to the medial malleolus Roentgenograph-

ically it can be visualized . . optimally with a very minimal amount of external rotation" *

It is questionable whether *os vesalianum,* as described by Vesalius in the sixteenth century, is a true accessory bone It usually conforms to the shape of the proximal end of the fifth metatarsal and often fuses with the metatarsal during puberty, forming the tuberosity of the metatarsal Baastrup (1921-1922) concluded that this bone is usually an accessory ossification center of the base of the fifth metatarsal (Fig 16)

Trolle (1948), whose series of 250 cases included accessory bones of the foot in the embryo and the full-term stillborn, thought that the following reasons account for their occurrence. (1) an independent ossification anlage, (2) fusion of more than one independent ossifying center, (3) ossification after birth brought about by external local stimuli, (4) an unidentified localized pathologic process Trolle emphasized that one and the same type of accessory bone need not have the same genesis in different persons.

Accessory sesamoids may be found under any or all of the metatarsal heads, or under the head of the proximal phalanges The constant accessory bones and sesamoids may produce painful symptoms

Ligaments of Foot and Ankle

The six ligaments of the foot and ankle are the deltoid, the anterior talofibular, the posterior talofibular, the calcaneofibular, the interosseous talocalcaneal, and the plantar calcaneonavicular

Deltoid Ligament.—The deltoid ligament (Fig 23) helps the stability of the ankle It is a strong, flat, fan-shaped band attached to the medial malleolus It consists of two sets of fibers superficial and deep Of the superficial fibers, the anterior pass forward to be inserted into the tuberosity of the navicular bone The middle fibers descend almost perpendicularly to be inserted into the sustentaculum The posterior fibers pass backward and are attached to the inner side of the talus The deep fibers are attached to the tip of the medial malleolus and to the medial surface of the talus

Anterior Talofibular Ligament—The anterior talofibular ligament passes from the anterior margin of the fibular malleolus forward and medially to the talus in front of its lateral articular facet

Posterior Talofibular Ligament.—The posterior talofibular ligament which rigidly binds the fibula and talus runs almost horizontally from the depression at the medial and back part of the fibular malleolus to a prominent tubercle on the posterior surface of the talus

Calcaneofibular Ligament.—The calcaneofibular or medial ligament is a narrow rounded cord extending from the apex of the fibular malleolus downward and slightly backward to a tubercle on the lateral surface of the calcaneus

*From March, H C, and London, R I Os Sustentaculi, Am J Roentgenol 76 1114-1118, Dec, 1956 Courtesy Charles C Thomas, Publisher

accessory bone (Pirie's bone) on the dorsum of the talonavicular (Fig. 21), and he also observed that this ossicle is generally mistaken for fracture Years later Gottlieb and Beranbaum (1950) made the same observation

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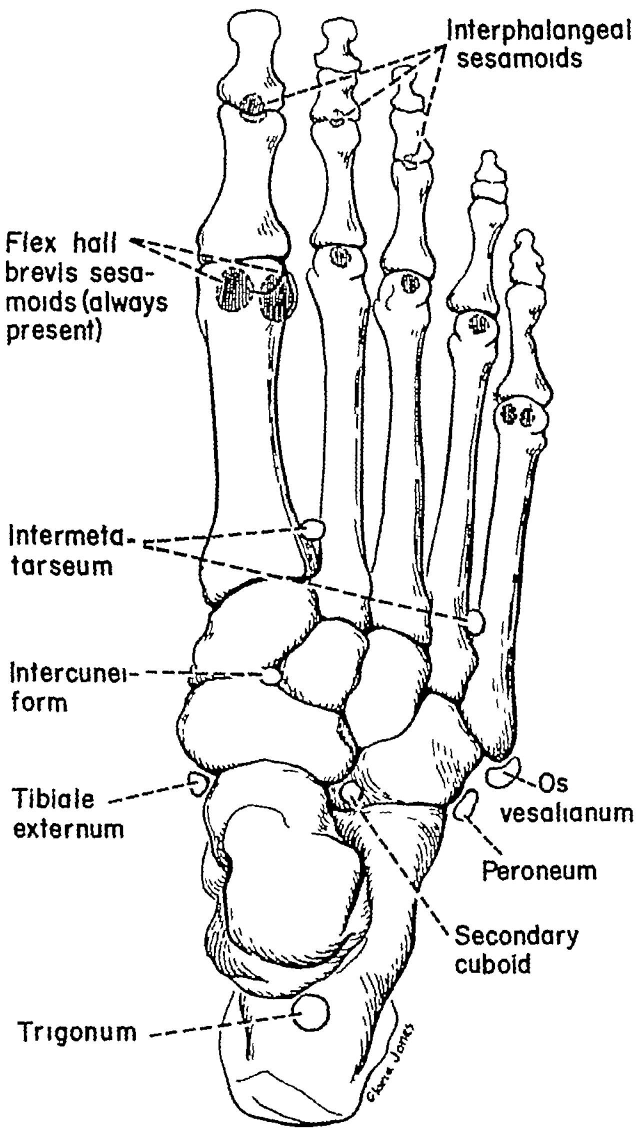


Fig 22 —Schematic drawing of accessory bones

as observers who regard "its existence as doubtful" March and London, however, encountered two cases within two weeks and added their reports They describe the ossicle as appearing "at the posterior end of the sustentaculum tali" and they report that it "produces a small visible and palpable projection just below and very slightly posterior to the medial malleolus . Roentgenograph-

and divides near the heads of the metatarsal bones into five processes, one for each of the toes Each process divides into two collateral strata the superficial stratum inserts into the skin of the transverse sulcus which separates the toes from the sole, the deeper stratum divides into two slips, to encompass the side of the flexor tendons of the toes, and blends in with the transverse metatarsal ligament

The lateral and medial portions of the plantar fascia are thinner than the central piece and cover the sides of the sole of the foot

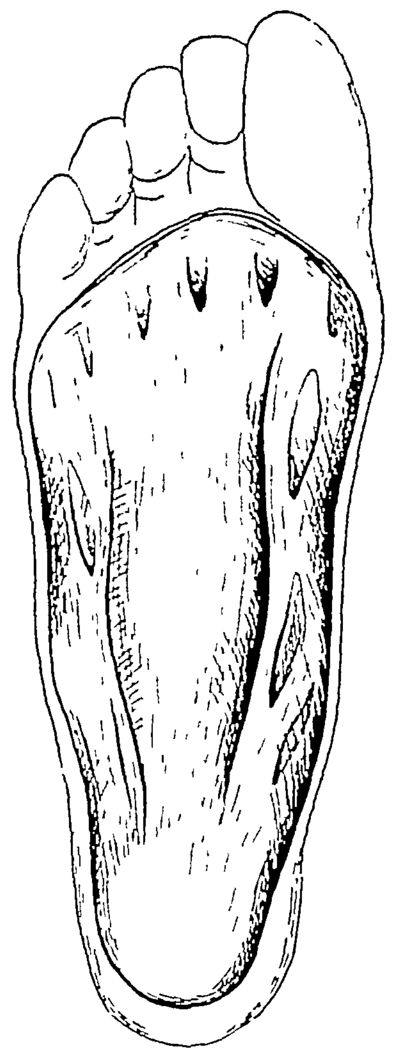


Fig 24 —Plantar aponeurosis Note narrow origin and wide insertion of plantar fascia which acts as a bowstring to longitudinal arch

The plantar fascia acts as a bowstring of the longitudinal arch and supports all the structures on the plantar surface

Muscles of the Foot

Dorsum. Extensor Digitorum Brevis.—The extensor digitorum brevis is the only muscle on the dorsum of the foot This muscle arises from the superior and lateral surfaces of the calcaneus and passes obliquely across the toe dorsum

Interosseous Talocalcaneal Ligament.—The interosseous talocalcaneal ligament which is thick and strong forms the chief bond of union between the two bones, the calcaneus and the talus, and reinforces the anterior and posterior talocalcaneal ligaments It lies in the space formed by the inferior surface of the neck of the talus and superior surface of the neck of the calcaneus, called the *sinus tarsi* It is attached to the groove between the articular facets of the under surface of the talus and to a corresponding depression on the upper surface of the calcaneum

Plantar Calcaneonavicular Ligament.—The plantar calcaneonavicular ligament is a broad and thick band of fibers which connects the anterior margin of the sustentaculum tali to the plantar surface of the navicular and supports the head of the talus

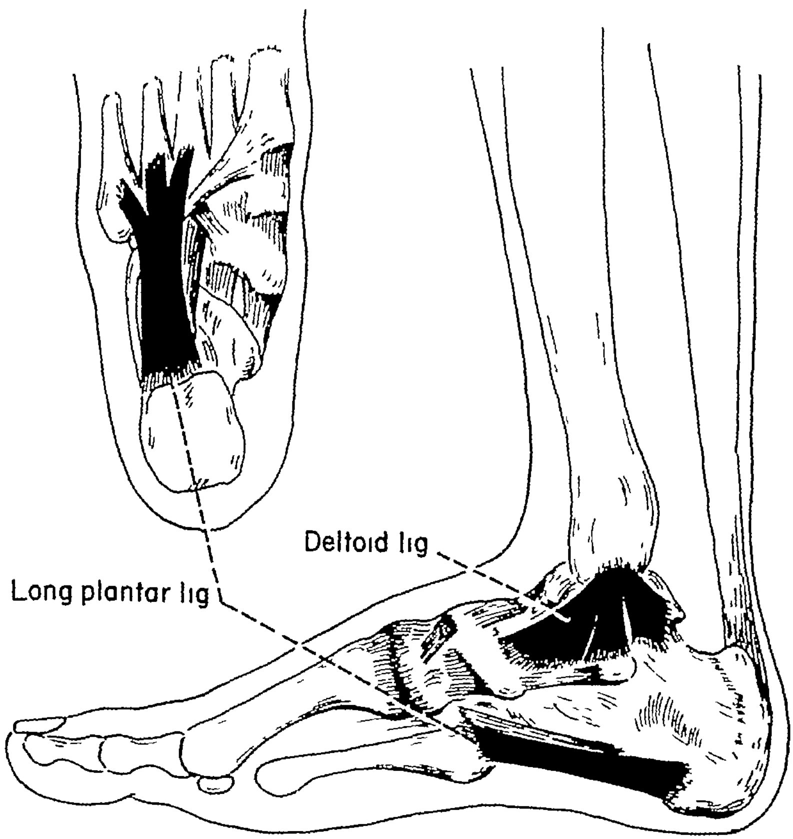


Fig 23 —Deep ligaments of foot and ankle

The Plantar Aponeurosis

The plantar aponeurosis (Fig 24) is a specialized fascia (plantar fascia) composed of dense white fibers running longitudinally from heel to toes It originates at its narrowest part from the tuberosity of the calcaneus, and, extending longitudinally, spreads out on the plantar surface of the foot It is divided into well-differentiated central, lateral, and medial parts

The central portion, the thickest and strongest, originates from the medial process of the calcaneal tuberosity, broadening and thinning as it passes forward,

from the fibular border of the inferior surface of the calcaneus and is inserted into the fibular margin of the tendon of the flexor digitorum longus muscle. The four lumbricales are small accessory muscles to the tendons of the flexor digitorum longus from which they arise. They are inserted on the tibial side of the base of the four lesser proximal phalanges.

Third Layer (Fig 25, III)—The *flexor hallucis brevis* arises as a broad process from the tibial side of the undersurface of the cuboid and from the third cuneiform It divides into two tendinous parts, which are inserted into the plantar surface of the base of the proximal phalanx of the great toe; a sesamoid is incorporated in each tendon at about 1.5 cm. proximal to the point of insertion of the tendon.

The *adductor hallucis* arises by two heads, oblique and transverse. The oblique head crosses the foot obliquely and arises from the bases of the second, third, and fourth metatarsal bones The transverse head arises from the plantar metatarsophalangeal ligaments of the third, fourth, and fifth toes and from the transverse metatarsal ligament. Their tendons conjoin under the first metatarsal interspace and are inserted into the fibular side of the base of the proximal phalanx of the great toe.

The *flexor digiti quinti brevis* lies under the shaft of the fifth metatarsal It arises from the base of the fifth metatarsal and is inserted into the fibular side of the base of the proximal phalanx of the fifth toe.

Fourth Layer—The four *interossei dorsalis* are located between the metatarsal shafts—all arising by two heads from the adjacent sides of the metatarsal bones—and are inserted into the bases of the proximal phalanges The three *interossei plantaris*, all single-headed (Fig. 25, *IV*), lie beneath the metatarsal bones They arise from the bases and tibial sides of the bodies of the third, fourth, and fifth metatarsal bones and are inserted by small rounded tendons into the tibial sides of the bases of the proximal phalanges of the same toes.

Blood Vessels of the Foot

Arteries.—The *dorsalis pedis* is a continuation of the *anterior tibial artery.* It passes forward from the ankle joint along the dorsum of the foot to the base of the first intermetatarsal space where the *dorsal arcuate branch* arises which supplies the fibular side of the second toe and both sides of the third, fourth, and fifth toes Very near, arise the *deep plantar artery* and the *first dorsal metatarsal artery* supplying the great toe

The *posterior tibial artery* begins at the lower border of the popliteus and extends obliquely downward, lying behind the tibia, coursing downward midway between the medial malleolus and the medial process of the calcaneal tuberosity where the posterior tibial artery divides beneath the origin of the abductor hallucis into the *medial* and *lateral plantar* arteries.

The *medioplantar artery* (Fig 26), which is smaller than the lateral, passes forward along the medial side of the foot, its three small branches pass to the cleft between the medial four toes and terminate in the metatarsal branches of the plantar arch

of the foot, dividing into four tendons One tendon inserts into the dorsal aspect of the first phalanx of the great toe The other three insert into the tendons of the extensor digitorum longus of the second, third, and fourth toes

Plantar: Four Layers of Muscles.—The muscles of the plantar surface of the foot are divided into four layers The first layer includes the abductor hallucis, the flexor digitorum brevis, and the abductor digiti quinti. The second layer consists of the lumbricales and the quadratus plantar muscle The third layer includes the flexor hallucis brevis, adductor hallucis, and flexor digiti quinti brevis The interosseous muscles, four dorsal and three plantar, make up the fourth layer

First Layer (Fig 25, I)—The *abductor hallucis* lies along the tibial plantar border of the foot covered by the medial border of the plantar fascia. It arises from the medial process of the tuberosity of the calcaneus. Its fibers end in a wide tendon which is inserted into the tibial side of the base of the proximal phalanx of the great toe

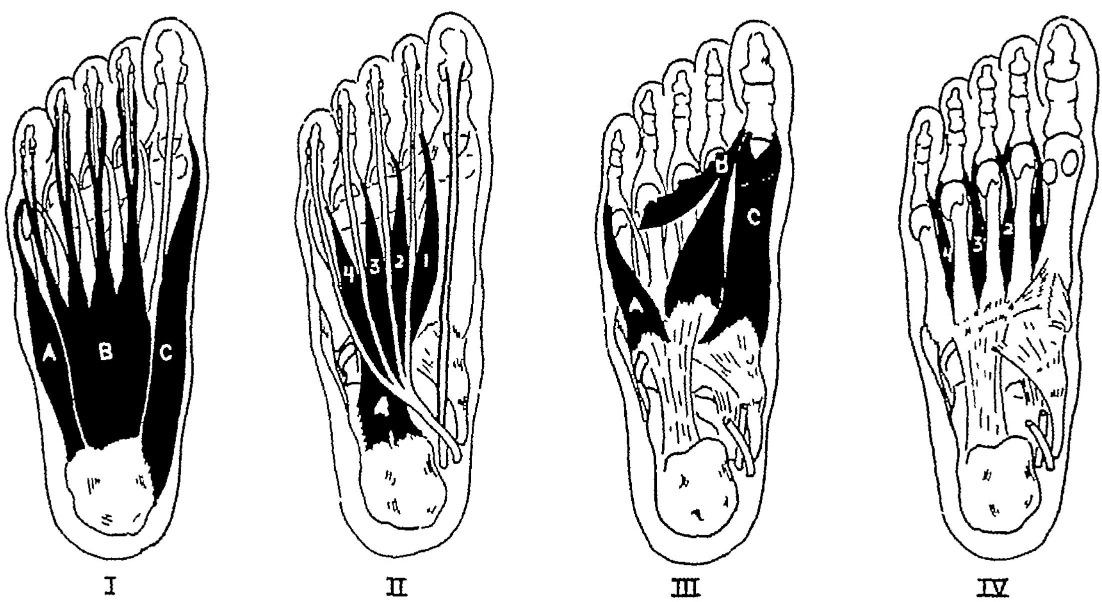


Fig 25—Layers of muscles on plantar surface of foot *I, A,* Abductor minimi digiti, *B,* flexor digitorum brevis, *C,* abductor hallucis *II, A,* Quadratus plantae, *1-4,* lumbricales *III, A,* Flexor brevis minimi digiti, *B,* adductor hallucis, *C,* flexor hallucis brevis *IV, 1-4,* Plantar interossei

The *flexor digitorum brevis* occupies the middle line of the sole of the foot immediately underneath the central part of the plantar fascia It originates by a narrow tendon from the tuberosity of the calcaneus and passes forward, dividing into four tendons which are inserted into the sides of the middle phalanx of the four smaller toes

The *abductor digiti quinti* lies along the lateral border of the foot, arising from the lateral process of the tuberosity of the calcaneus and running forward along the fibular side of the sole It is inserted, together with the flexor digiti quinti brevis, into the fibular side of the base of the first phalanx of the fifth toe

Second Layer (Fig 25, II)—The quadratus plantar is divided into two parts, separated from each other by the long plantar ligament It arises as a flat tendon

The *lateroplantar artery,* which is larger than the medial, passes obliquely lateralward and across the sole and forward to the base of the fifth metatarsal bone It then bends medialward to the interval between the bases of the first and second metatarsal bones where it unites with the deep terminal plantar branch of the dorsalis pedis artery, which completes the plantar arch

The *plantar arch,* in addition to distributing numerous branches to the muscles of the sole, gives off the *perforating artery* and *plantar metatarsal branches.*

Veins.—The important veins of the foot bear the same names as the arteries They follow the course of the arteries except for the medial superficial marginal veins (Fig. 27), from which originate the long saphenous vein, and the lateral marginal vein, from which originates the small saphenous vein

Nerves of the Foot

The nerves of the foot are the medioplantar, the digital, the lateroplantar, the dorsal deep peroneal, and the mediodorsal cutaneous (Fig 28).

The *medioplantar nerve* (Fig. 29), the larger of the two terminal divisions of the tibial nerve, accompanies the medioplantar artery From its origin under the laciniate ligament, it passes forward and laterally under cover of the abductor hallucis to the interval between this muscle and the flexor digitorum brevis, and gives off a proper digital plantar nerve It then divides opposite the bases of the metatarsal bones into three common digital plantar nerves and a proper digital nerve of the great toe

The three common *digital nerves* include those of the first common digital nerve which supply the adjacent sides of the great and second toes, those of the second, supplying the adjacent sides of the second and third toes, and those of the third, supplying the adjacent sides of the third and fourth toes

The *lateroplantar nerve* accompanying the lateroplantar artery supplies the skin of the fifth toe and lateral half of the fourth toe

The *dorsal deep peroneal nerve* begins at the bifurcation of the common peroneal nerve It descends with the anterior tibial artery to the front of the ankle joint where it divides into a lateral and a medial terminal branch The lateral terminal branch passes across the tarsus and supplies the tarsal joints and the metatarsophalangeal joints of the second, third, and fourth toes The medial terminal branch divides into two digital nerves which supply the adjacent sides of the great and second toes

The mediodorsal *cutaneous nerve* divides into two dorsal digital branches, one of which supplies the medial side of the great toes, the other, the adjacent side of the second and third toes The intermediate dorsal cutaneous nerve supplies the adjacent sides of the third and fourth toes and of the fourth and fifth toes

Surgical Landmarks

For surgical requirements the following structures should be studied minutely great toe joint, first metatarsal head; adductor hallucis, flexor hallucis longus and flexor hallucis brevis, base of fourth metatarsal in relation to tarsus,

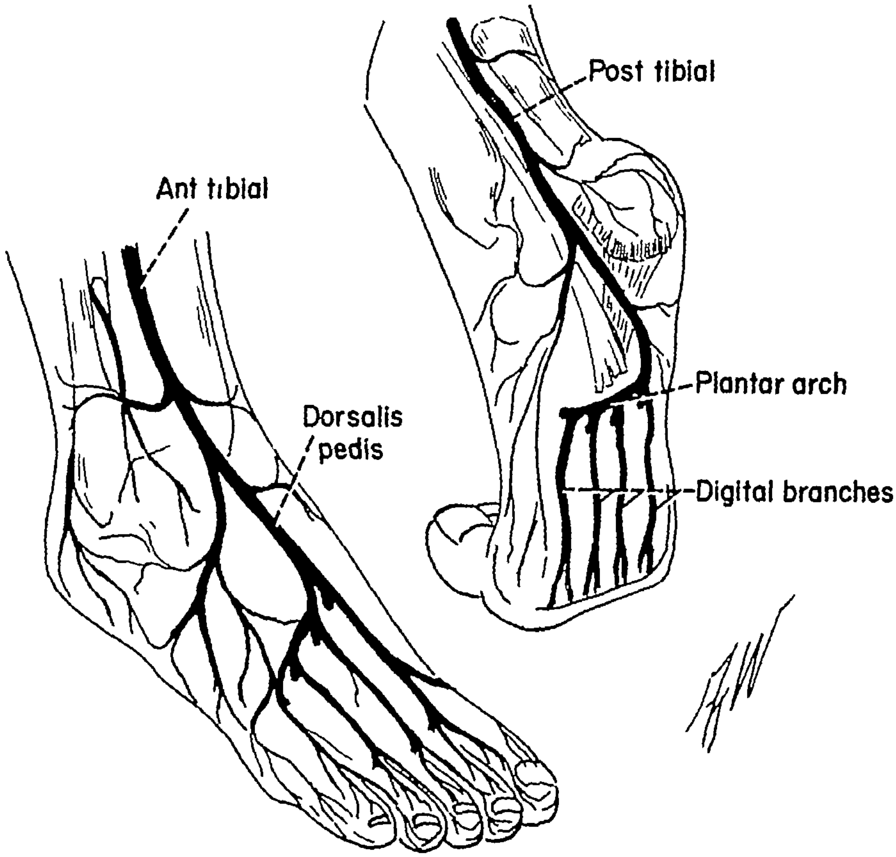


Fig 26 —Arteries of foot

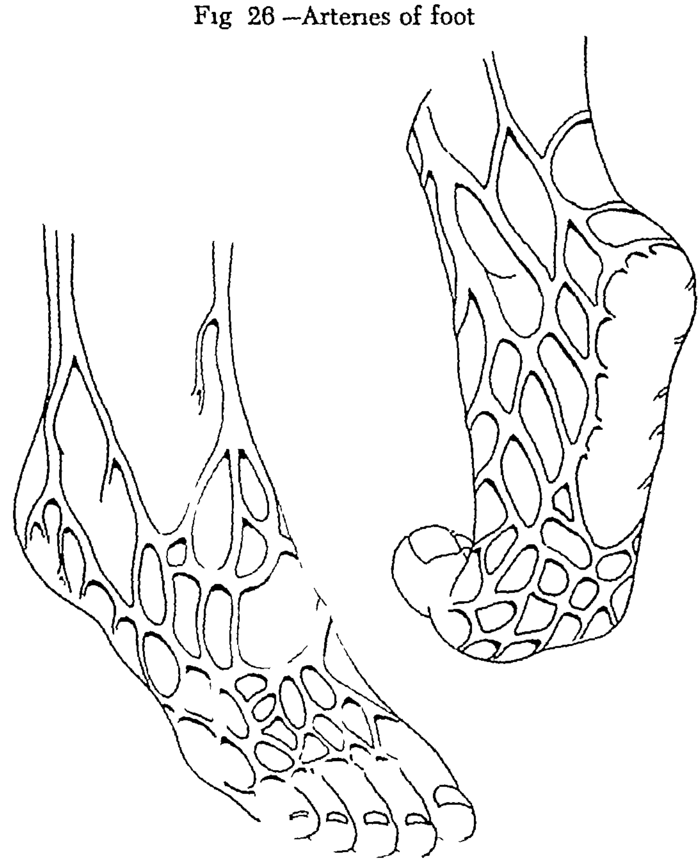
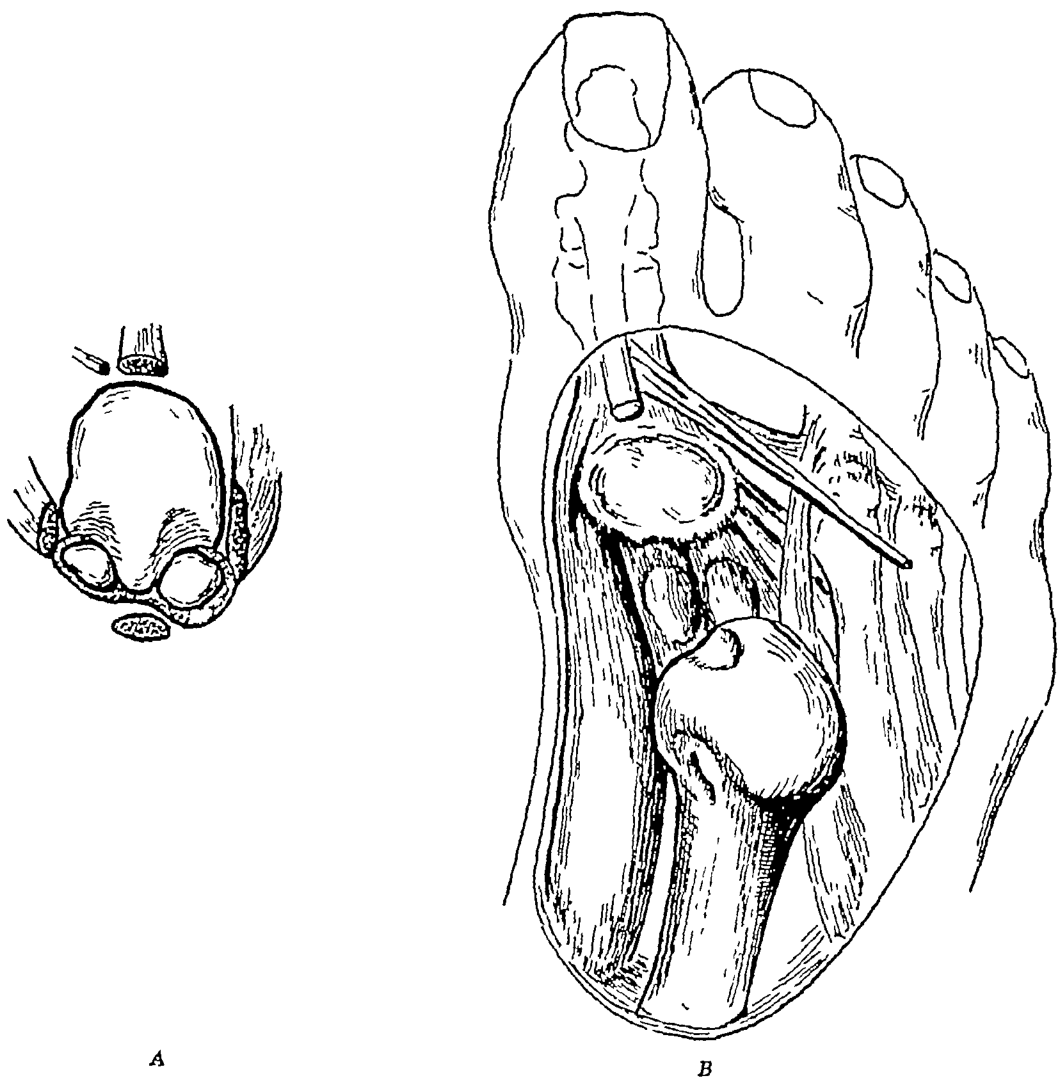


Fig 27 —Superficial veins of foot

base of fifth metatarsal; deltoid ligament, posterior surface of calcaneus, retrocalcaneal bursae, accessory bones.

Great Toe Joint.—The great toe joint is the most complex joint in the foot. It is the most important weight-bearing and balancing part of the forefoot The function of this joint is controlled by two muscles of the leg, the *extensor* and *flexor hallucis longus,* which are inserted into the distal phalanx, and by four intrinsic foot muscles, the abductor hallucis and adductor hallucis, the flexor hallucis brevis, and the extensor hallucis brevis which is a tendon filament from the extensor digitorum brevis, all are inserted into the base of the proximal phalanx and provide the versatile movement of the great toe The hallux sesamoids are incorporated in the tendon of the flexor hallucis brevis and lie directly under the head of the first metatarsal (Fig. 30). Any error in these structures influences deformity of the great toe joint

First Metatarsal Head.—The plantar surface of the first metatarsal head has two sagittal sulci for articulation of the sesamoids (Fig 30). A deep groove



A *B*

Fig 30—*A,* Cross section through first metatarsophalangeal joint showing relation of sesamoids and tendons to first metatarsal head *B,* Dorsal exposure of first metatarsophalangeal joint with toes in plantar flexion

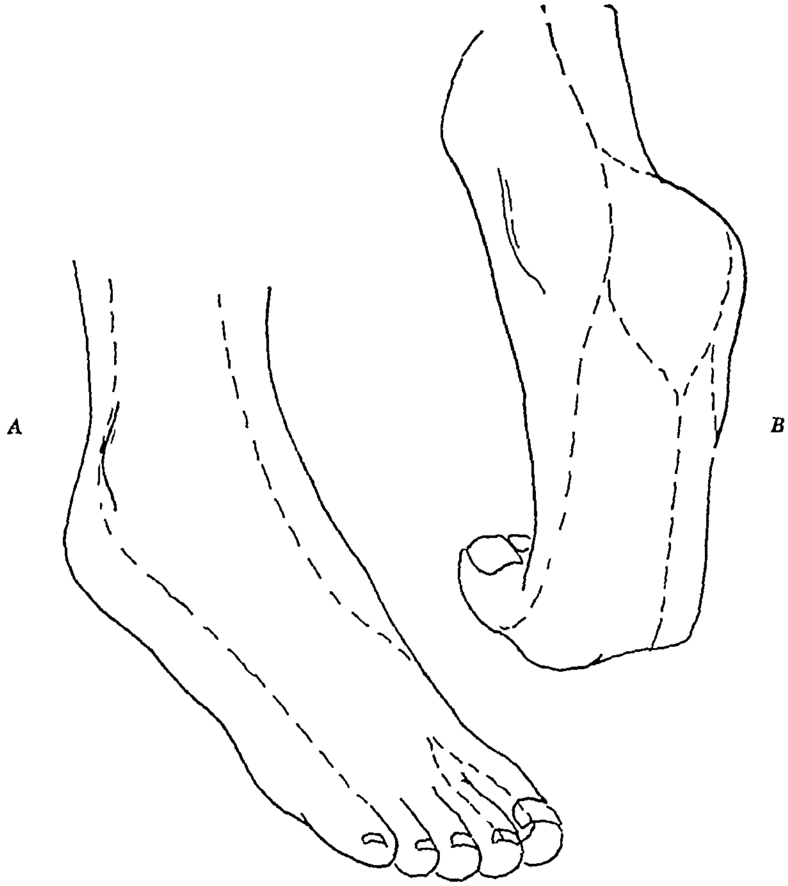


Fig 28 —Segmental distribution of cutaneous nerves of foot A, Dorsum B, Plantar

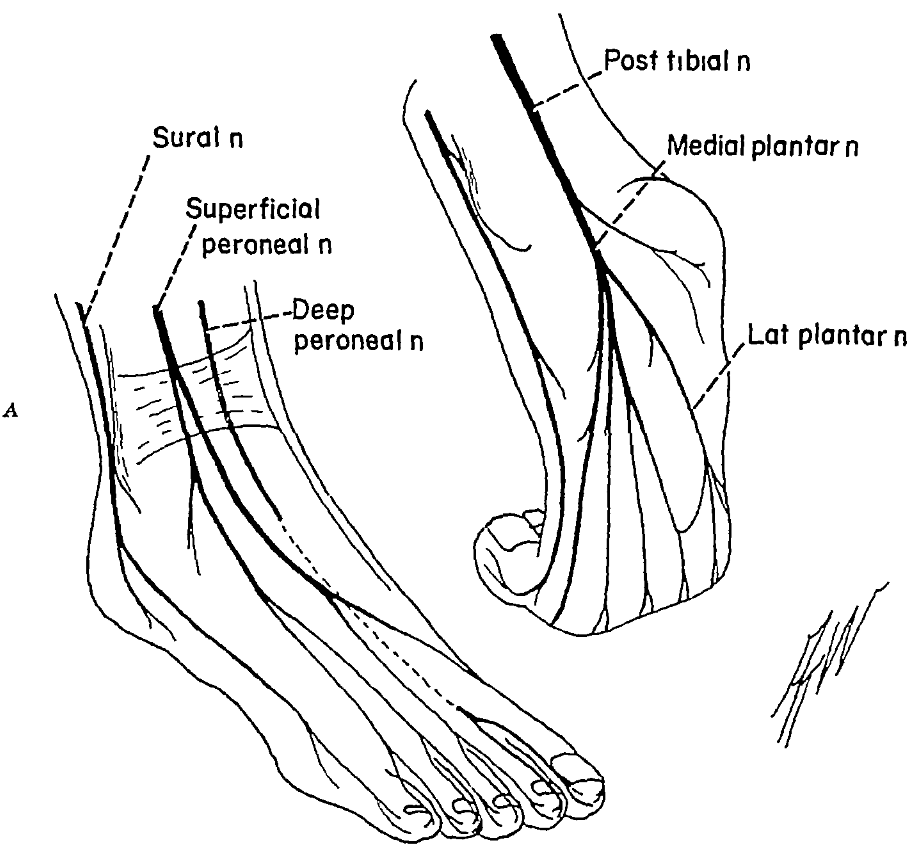


Fig 29 —Deep nerves of the foot A, Dorsum B, Plantar

Base of the Fifth Metatarsal.—The base of the fifth metatarsal acts as the adjacent bone to the keystone's position of the cuboid (Fig 33). It also forms the side of the sulcus which contains the tendon of the peroneus longus Disease, injury, or deformity of the base of the fifth metatarsal bone can produce disability

Deltoid Ligament.—The deltoid ligament is a thick, strong, triangular-shaped ligament which helps stabilize the medial side of the ankle and maintain the longitudinal arch

Posterior Surface of Calcaneus.—The posterosuperior surface of the calcaneus is normally convex but may vary from straight to convex (Fig 34) Such a variable may become a point of irritation because the standard shoe counter is convex

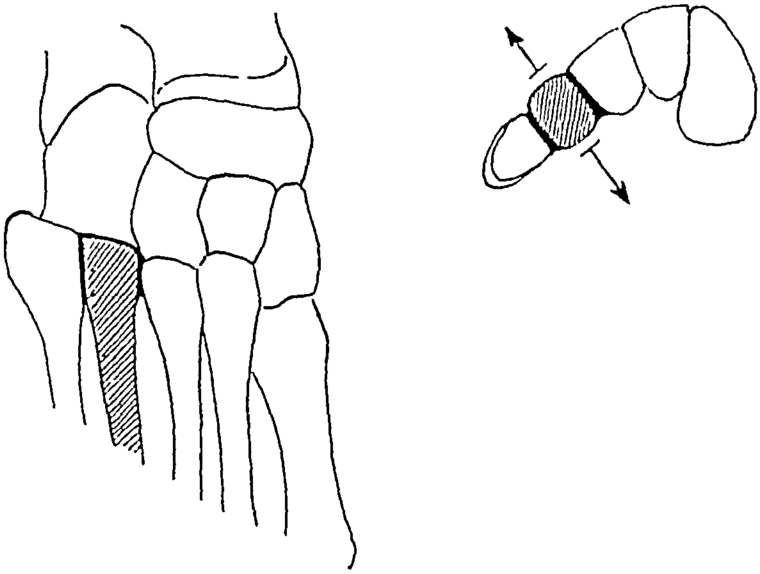


Fig 32—Normal articulation of base of fourth metatarsal Arrows in small sketch point to normally unstable articulation of base of fourth metatarsal

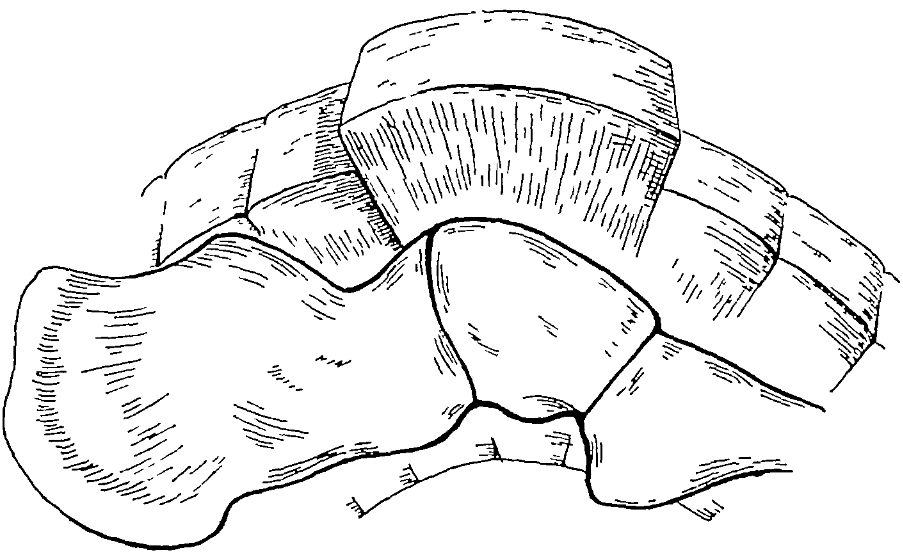


Fig 33—Keystone structure of cuboid which holds base of fifth metatarsal and head of calcaneus in position

on the articular surface of the head, about 2 mm from the tibial side, separates the condylar process from the head of the first metatarsal Deformities of the first metatarsophalangeal joint are modified or accentuated by variations in the contour of the first metatarsal head

Adductor Hallucis—The adductor hallucis, sometimes called the *lobster-claw muscle*, is more powerful than its opponent, the abductor hallucis Because it is inserted into the fibular side of the base of the proximal phalanx of the great toe, the adductor hallucis participates in pulling the base of the proximal phalanx off the head of the first metatarsal (Fig 31), as seen in hallux valgus

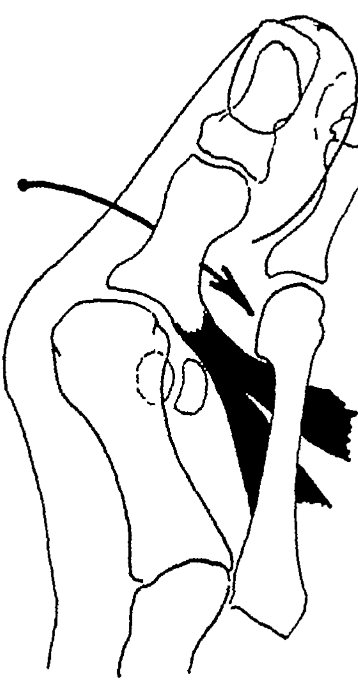


Fig 31 —Arrow shows direction of pull of adductor hallucis

Flexor Hallucis Brevis.—The flexor hallucis brevis has its broad tendon containing the hallux sesamoids, the muscle is inserted into the base of the first proximal phalanx and controls the plantar flexion at the first metatarsophalangeal joint

Base of Fourth Metatarsal in Relation to Tarsus.—Of the five metatarsals the base of the fourth metatarsal articulates less securely with the tarsus than do the other four (Fig 32) The base of the first metatarsal articulates by a large surface with the first cuneiform and is fixed by the tendons of the tibialis anticus, tibialis posticus, and peroneus longus The base of the fifth metatarsal is a weight-bearing point fixed by the tendon of the peroneus brevis The bases of the second and third metatarsals are wedged like keystones at their tarsal-metatarsal articulation The base of the fourth metatarsal has no other fixation than the fascial components around it, as a result, the entire bone has a slight floating motion which may account for so many common disorders at the distal end of the fourth metatarsal bone (Morton's toe)

tional space a deep space between the extenso digitorum brevis and the tarsal bones

The plantar fascial spaces require further delineation.

Central.—The *first* central plantar space is beneath the center of the plantar aponeurosis, between the aponeurosis and the flexor digitorum brevis, extending from the midtarsal joint to almost the middle of the first metatarsal The *second* plantar space is between the flexor digitorum longus tendons with their assisting lumbricales muscles and the flexor digitorum brevis The *third* central plantar space is deeper than the second in the outer proximal aspect but is also between the flexor digitorum longus (and their accompanying lumbricales) and the oblique adductor hallucis, the second and third plantar, and third and fourth interossei The *fourth* central plantar space is deepest It is beneath the adductor hallucis oblique muscle, the shape of which it follows, and is located over the first and part of the second and third metatarsals and their interossei.

Fibular.—Of the two fibuloplantar spaces, one is located under the proximal part of the fifth metatarsal bone, deep under the flexor digiti brevis, and the other, at the deeper aspect and origin of the abductor digiti quinti between that muscle and its fascia, extending almost to the fifth metatarsal bone

Tibial.—One tibioplantar space is deep between the abductor hallucis and its fascial lining, it begins at the ligamentum laciniatum and extends to the point where this muscle merges with its tendon at the posterior third of the first metatarsal

FUNCTION

The outer part of the human foot is specialized for static weight transmission, the inner part, which is elastic and mobile, is specialized for propulsion By lying flat on the ground the outer border of the foot forms a buttress for stability and balance, the inner portion is essentially a lever

The foot performs static and kinetic functions Its static function is in support of the body, its kinetic function is in providing leverage for propulsion The foot acts as a shock absorber on impact and when the body is in motion, it propels the body in walking, climbing, descending, jumping, and dancing

Turchin (1955) interprets the purpose of the foot as a transmitting mechanism to a supporting structure rather than as being a supporting structure in itself, the basic support is the surface upon which we stand or walk A supporting surface must be capable of displacing the weight of the body, hence, man cannot walk on water or air The pressure felt on the foot while standing or in motion is not exerted by the body's weight but is produced by the resistance of the ground, which is the opposite-force at the point of contact with the transmitting structure It is reasonable, therefore, to consider the foot primarily as a transmitter to a supporting structure Different supporting structures exert different resistances to the feet, as explained by Newton's Third Law *For every action there is an equal and opposite reaction* A cork floor or soft ground, having greater resilience than a concrete floor, can exert displacement pressure over a larger area of the foot Feet that must perform their functions on a nonconform-

Hallux Sesamoids.—Hallux sesamoids vary in size, shape, and position, so that at times the sesamoids become sharp pressure points, or they may join in accentuating deformities of the first metatarsophalangeal joint.

Retrocalcaneal Bursae.—The only consistent anatomic bursae of the foot are the retrocalcaneal bursae They lie on the posterosuperior surface of the calcaneus and between the posterior surface of the talus and anterior surface of the tendo achillis (Fig 35)

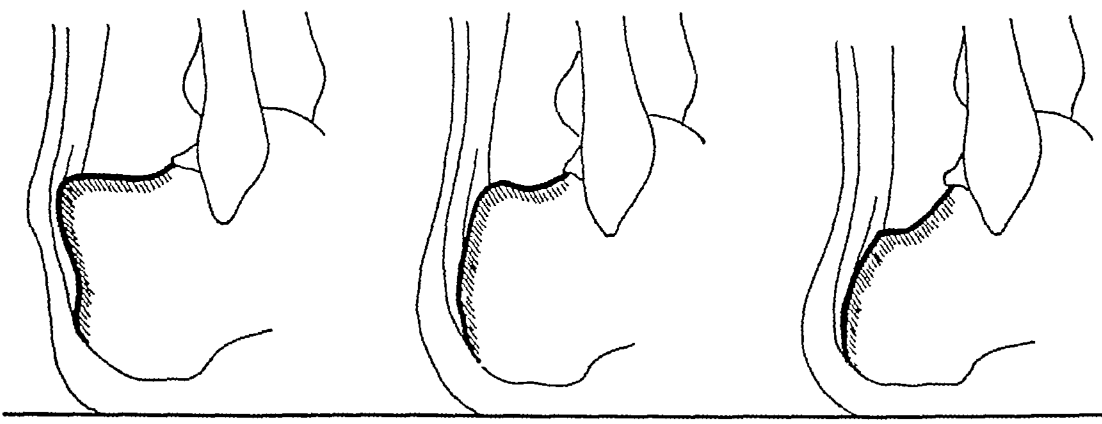


Fig 34.—Variations in shape of superior tuberosity of calcaneus *A*, Hyperconvex (so-called Haglund's disease) *B*, Normal *C*, Hypoconvex

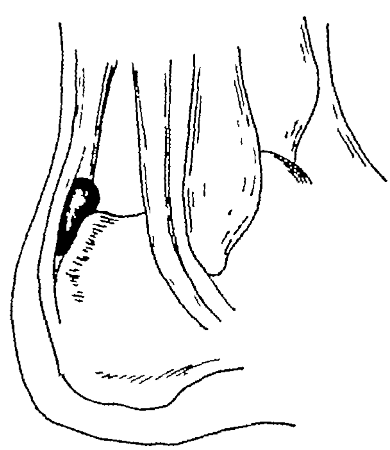


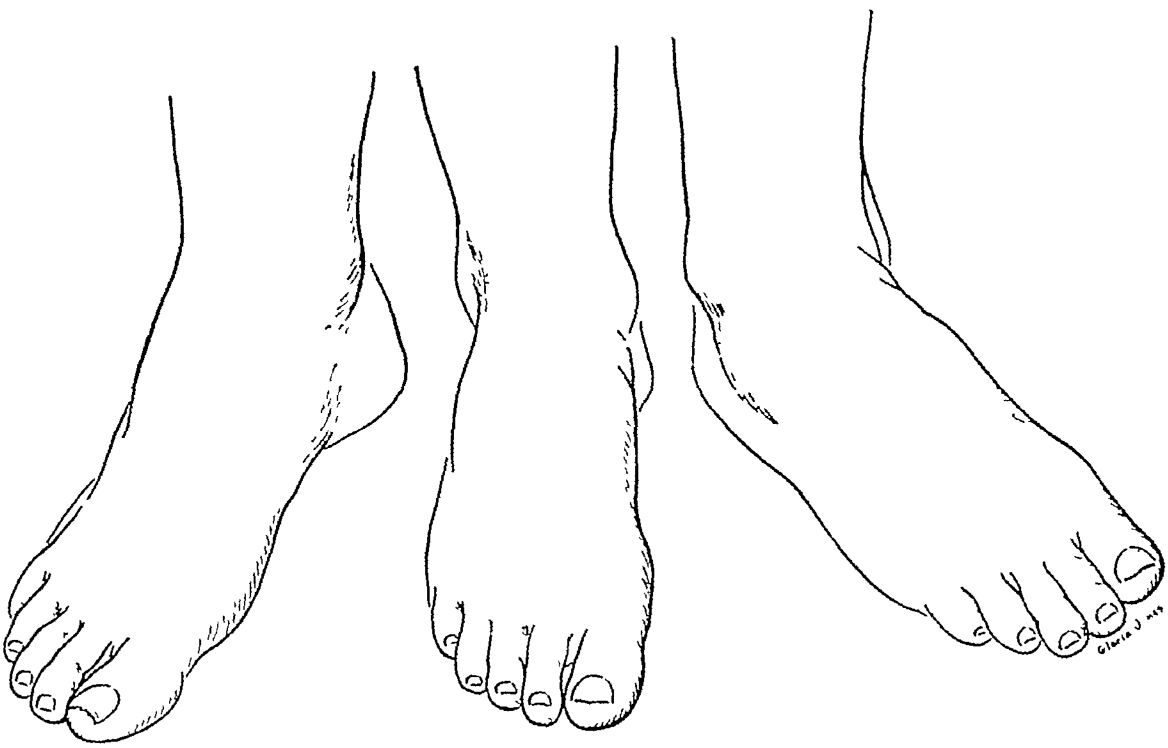
Fig 35.—Retrocalcaneal bursa located between the tendo achillis and superior tuberosity of calcaneus

Fascial Spaces.—Rao and Kini (1933) have described the fascial spaces of the foot comprehensively and in terms of their boundaries, floors, and roofs To recapitulate, there are three fascial plantar spaces, divided by septa into four median, two lateral, and two medial spaces There are two dorsal spaces one is a subcutaneous continuation of leg space and the other, subaponeurotic, extends between deep fascia across the tarsals and metatarsals Four lumbrical spaces, four interdigital subcutaneous spaces, and four web spaces, one for each interdigital cleft, account for the remaining fascial spaces of the foot, with one addi-

> pination, as are eversion and abduction in the single movement of pronation A further source of confusion is that inversion and eversion are often used loosely when supination and pronation are meant *

It is true that pronation and supination are functions generally ascribed to the foot, however, pronation and supination are rotary movements The source of rotation of the hand is mainly in the elbow, a similar motion of the foot would have had to originate in the knee. But this joint has no such rotary movements, therefore, it is incorrect to ascribe the functions of pronation and supination to the foot.

Leg and Foot Muscles.—All major movements of the foot are performed by muscles of the leg The intrinsic muscles of the foot are secondary in performance except those muscles inserted into the base of the first proximal phalanx They are essential to the function of the first metatarsophalangeal joint and also contribute adversely to the production and maintenance of static deformities of the foot, as in hallux valgus



A B C

Fig 37 —Positions of foot A, Abduction B, Normal C, Adduction

Ankle.—In plantar flexion and dorsiflexion the entire action is accomplished by the movement of the talus in the ankle mortise, as is eversion and inversion

Talonavicular Joint—The talonavicular joint is the most important joint in the movements of the forefoot Adduction and abduction are accomplished primarily by this joint

*From Shephard, Edmund Tarsal Movements, J Bone & Joint Surg 33B 258-263, May, 1951

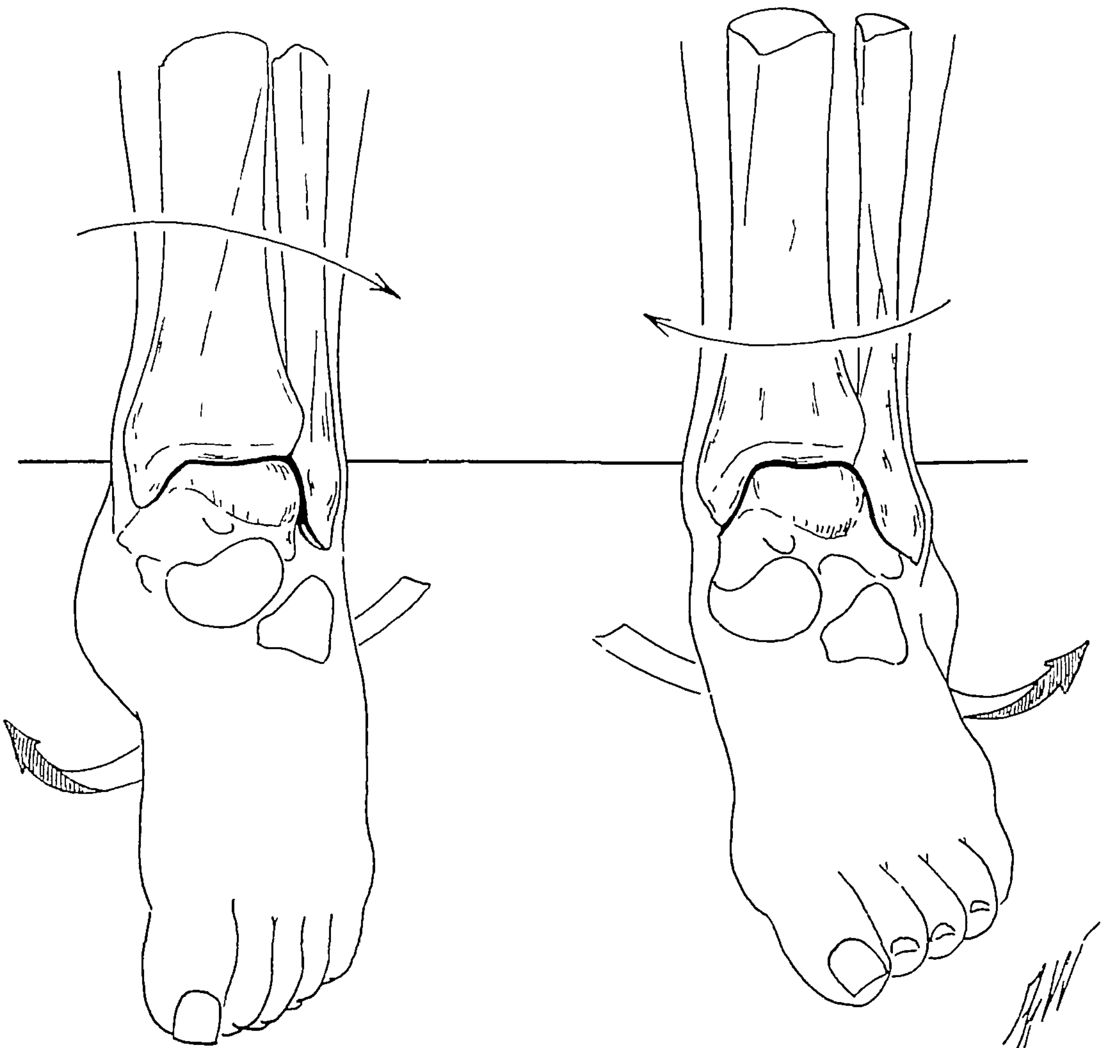
ing supporting structure, such as a concrete floor, are taxed beyond their architectural intention of functioning on a yielding supporting surface

Movements of the Foot

The primary movements of the foot are eversion and inversion (Fig 36), adduction and abduction (Fig 37), and dorsiflexion and plantar flexion (Fig 38)

Shephard (1951) has attempted a distinction between terms of reference to the movements of the foot

> Inversion and eversion (rotation of the foot about its long axis), adduction and abduction (rotation about a vertical axis) are sometimes considered incorrectly as isolated movements It has long been recognized that they cannot occur independently and that inversion and adduction are always combined in the single movement of su-



A B

Fig 36 —Movements of the foot A, Inversion B, Eversion

Each of the three divisions of the foot, tarsus, metatarsus, and phalanges, has its individual function determined by structural design.

Tarsus.—The seven blocklike bones of the tarsus, having mainly flat articular surfaces, provide only a limited gliding motion and no rotary movements That is why the only muscles inserted into this region are those which move the foot as a unit on the ankle

Metatarsals.—The five metatarsals have a gliding type of joint at their tarsal articulation, thus, motion in this region is limited. The distal ends of the metatarsals are round and have rotary movement controlled by muscles whose tendons are inserted into the phalanges

Phalanges.—The fourteen phalanges have a freely movable ball-and-socket type articulation with one another as well as with the metatarsal heads, hence, the profuse supply of muscle insertions into the phalanges to move the toes individually or collectively.

Great Toe.—The great toe *(hallux)* differs from the other toes in that it has no middle phalanx but has six separate and distinct muscles inserted into it, enabling a wide range of motion comparable to the range of the thumb Although the great toe has lost its prehensile function, it has retained the complex anatomy needed for prehensility. That is why in cases of congenital absence of the hands, the foot can be trained to perform the functions of the hand The hallux performs the function of propulsion as well as functions of gripping and holding, and it also assists importantly in the balance of the body through weight transfer of the foot.

Ligaments.—The numerous ligaments that bind the bones of the foot together maintain the normal relationships of the bones to one another Although the ligaments are fibrous and nonelastic, they are not meant to have a weight-transmitting function but rather the function of holding temporarily the bones of the foot while the body weight is transferred to the ground, hence, pain and symptoms of foot strain attend occupations that require long periods of standing (Keith, 1928) The ligaments cannot withstand unremitting stress, as enunciated in Davis' Law

> *Ligaments, or any soft tissue, when put under even a moderate degree of tension, if the tension is unremitting, will elongate by the addition of new material, on the contrary, when ligaments or other soft tissues remain uninterruptedly in a loose or lax state, they will gradually shorten as the effete material is removed*

Direction of Lines of Force.—All lines of force travel primarily in a straight line Body weight is normally transferred only through a straight line through the tibia to the talus to the forefoot When body weight *(force)* does not travel in a straight line, a strain is exerted at the point of attempted direction because there is no supporting structure to absorb the downward force That strain may result in a tear when the force has no plane upon which to transfer its power An example of this is the foot that turns in extreme adduction during propulsion, the line of force goes through the lateral side to the ankle, resulting in a sprained ankle If the force is severe the strain may tear the collateral ligaments or even fracture the lateral malleolus

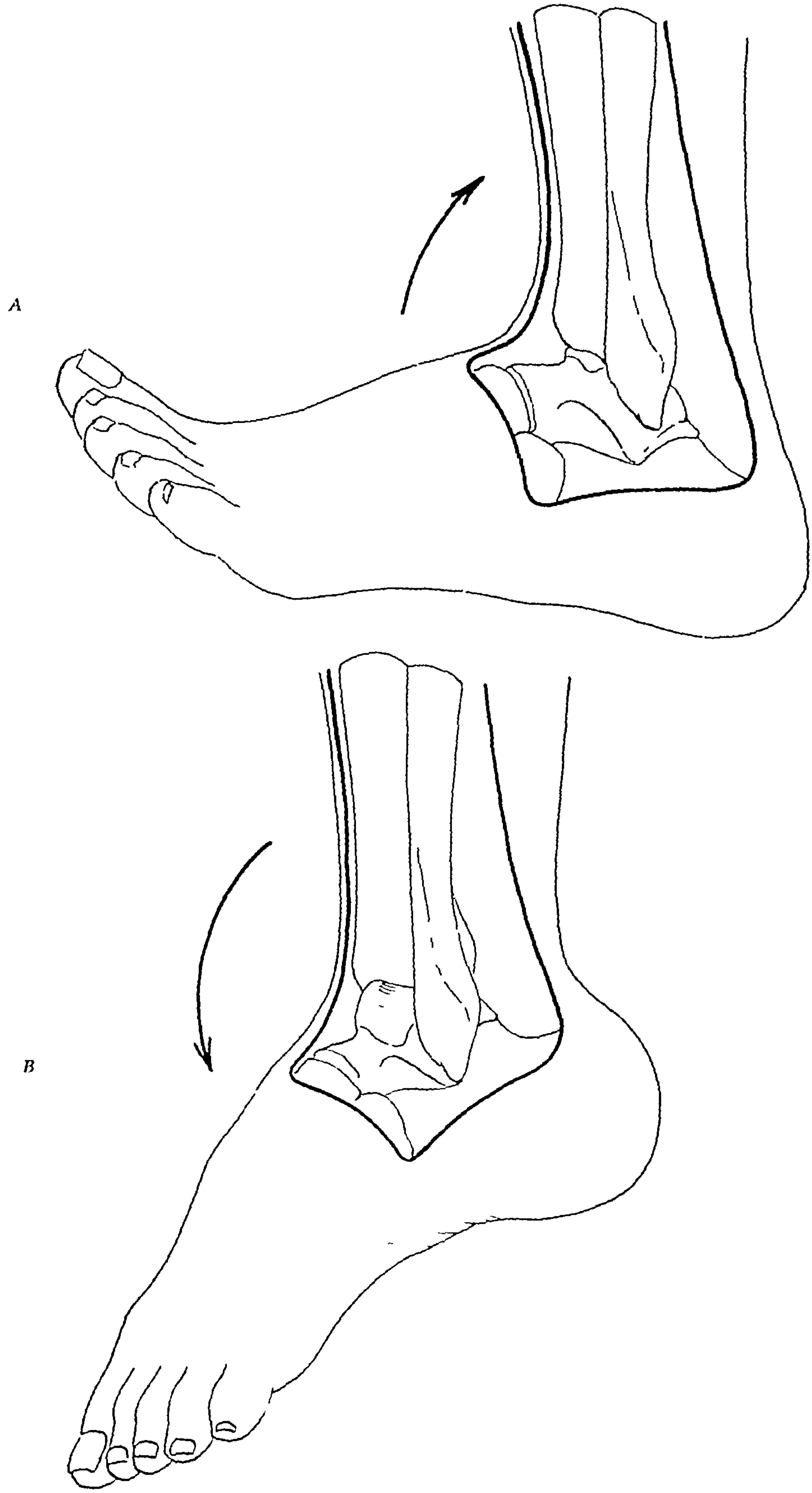


Fig 38 —Movements of the foot *A*, Dorsiflexion *B*, Plantar flexion

Static and Kinetic Functions

Standing (Static).—In standing, most of the body weight is transmitted directly to the talus, which distributes most of the weight to the calcaneus. The weight is then transmitted through the cuboid to the fifth metatarsal The remaining weight is transmitted from the head of the talus through the navicular and first cuneiform to the first metatarsal, forming a tripod on the plantar surface (Fig 39) The three middle metatarsals bear only a small amount of the body weight while standing

Walking (Kinetic)—Walking is essentially a falling motion Breaking of the fall with the opposite foot results in upright propulsion of the body The thrust of the body forward and the muscular action of the leg put the tarsal region in an upward angle from the heel while the weight is transferred throughout the metatarsals, from the base of the metatarsals through their heads (Fig 40), then the body is again in position for the next progressive falling motion—a complete step in walking In normal gait the weight of the body is transmitted through the talus to the calcaneus, transferred forward on the outer side of the foot, then the weight crosses over the heads of the metatarsal bones to the inner side of the foot and to the great toe The functions of the other toes are those of gripping and balancing

Shock Absorption in Propulsion—The four arches of the foot are formed in early fetal life and are a human peculiarity There are the inner longitudinal arch, the anterior transverse or metatarsal arch, the outer longitudinal arch, and the posterior transverse arch Only the inner longitudinal and the anterior transverse (or metatarsal) arches are significant

The *longitudinal arch* is the span or roof of bone over the sole which makes no contact with the ground when weight is borne on the supporting foot. It arches up from behind the tuberosity of the calcaneus, includes the intervening elements of the tarsus, and meets the floor again under the heads of the metatarsal bones The longitudinal arch, because of its high degree of resistance and elasticity, has the function of shock absorption during propulsion The plantar aponeurosis acts as a bowstring to this arch

The *transverse metatarsal arch* is formed by the five metatarsal heads, the first and fifth form the inner and outer pillars The transverse metatarsal ligament forms the bowstring for this arch The function of the transverse metatarsal arch is comparable to that of a cantilever spring as it absorbs the shock of body weight when it is transmitted to the metatarsal heads Whereas the longitudinal arch absorbs the initial shock and most of the shock of body weight during propulsion, all the component parts (the five metatarsal heads) of the transverse metatarsal arch do touch the ground during each step The first and fifth metatarsal heads meet the ground first, followed by the middle three The middle three metatarsal heads bear weight only a fraction of the time that the entire anterior arch touches the surface during propulsion

REFERENCES

Baastrup, I Os Vesalianum Tarsi and Fracture of Tuberositas Ossis Metatarso V, Acta radiol 1 334-348, 1921-1922

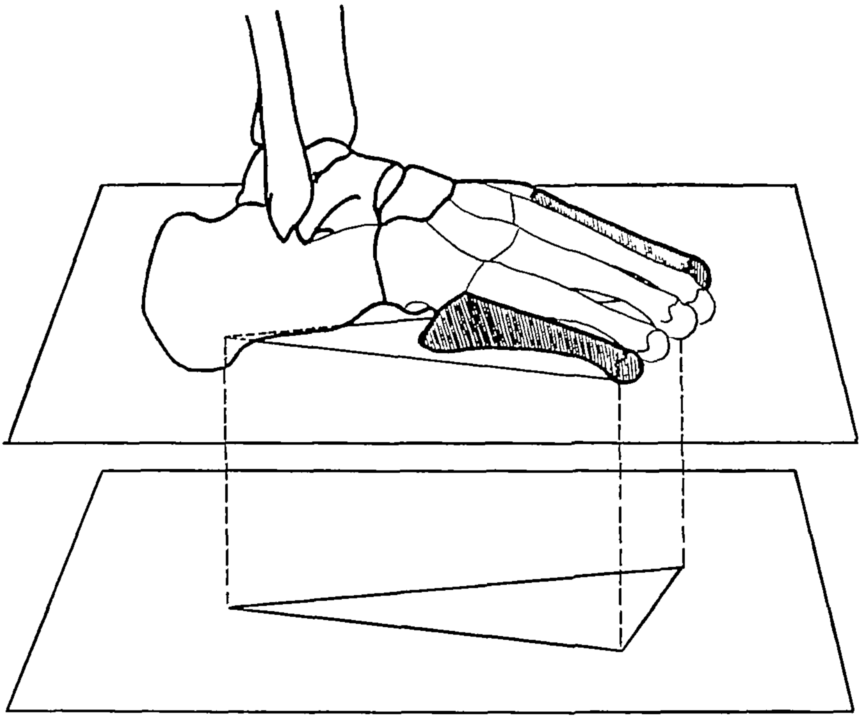


Fig 39 —Three-point weight transmission during standing Bottom triangle drawn for clearer representation of static weight transmission

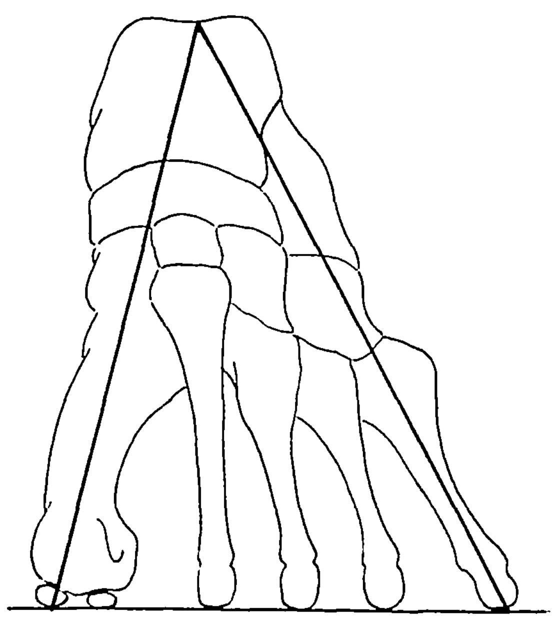


Fig 40 —Heavy lines show weight distribution to metatarsal heads

CHAPTER TWO

Examination and Diagnosis

DIAGNOSTIC ARMAMENTARIA

THE FOLLOWING AIDS ARE NECESSARY IN THE DIAGNOSIS OF FOOT DISABILITIES: the oscillometer, jewelers' eyeglasses, a skeleton of the normal foot, roentgenograms, pedographs

The *oscillometer* measures the extent or indicates the absence of arterial circulation Immediately above the ankle there is a normal index of between 2 and 4 degrees of oscillation In advanced cases of arterial occlusion the reading will be below one and may register at zero

Jewelers' eyeglasses help to identify minute changes that occur in the surface of the foot They are also useful visual aids to detection during palliative treatment of disorders of the toenails and when dissecting small deep-seated nucleated masses Jewelers' eyeglasses magnify details on roentgenograms not visible to the naked eye

A *skeleton of the normal foot* is helpful for comparison of the roentgenogram and the clinical disclosures made during examination

Roentgenograms are essential in the diagnosis of foot disabilities However, one must not lose sight of the two-dimensional limitation of an x-ray plate It is almost impossible to visualize some areas of the foot in two dimensions no matter how many views are taken The third dimension of a particular view may be entirely different from that which is disclosed in the roentgenogram A medial view of a calcaneal spur appears as a point of a pin, whereas actually the spur is about 2 cm wide from medial to lateral position Most medial or lateral views of the forefoot are distorted because the bones overlap

A *pedograph,* or imprint of the foot during weight bearing, normally shows a distinct outline and impression (Fig 41) Deviations from the normal may appear at points of increased pressure Areas of decreased density may be due to favoring of the foot because of pain or faulty weight distribution

Close, J R Some Applications of the Functional Anatomy of the Ankle Joint, J Bone & Joint Surg 38A 761-781, July, 1956
Close, J R, and Inman, V T Action of the Ankle Joint, Prosthetic Devices Research Project, Institute of Engineering Research, University of California (Berkeley) Report to the Advisory Committee on Artificial Limbs, National Research Council, Series 11, No 22, 1952
Duchenne, G B A Physiology of Motion Demonstrated by Means of Electrical Stimulation and Clinical Observation and Applied to the Study of Paralysis and Deformities, Philadelphia, 1949, J B Lippincott Co
Gottlieb, Charles, and Beranbaum, S L Pirie's Bone, Radiology 55 423-424, Sept, 1950
Grant, J C B An Atlas of Anatomy, ed 2, Baltimore, 1947, Williams & Wilkins Co
Gray, Henry Anatomy of the Human Body, ed 26, Philadelphia, 1954, Lea & Febiger
Jones, F W Structure and Function as Seen in the Foot, Baltimore, 1944, Williams & Wilkins Co
Jones, L Human Foot. Experimental Study of Its Mechanics and Role of Its Muscles and Ligaments in Support of Arch, Am J Anat 68 1-39, Jan, 1941
Keith, A History of the Human Foot and Its Bearing on Orthopedic Practice, London, 1928, H O Thomas Memorial Lecture
March, H E, and London, R I Os Sustantaculi, Am J Roentgenol 76 1114-1118, Dec, 1956
Morton, D F The Human Foot, New York, 1935, Columbia University Press
Pirie, A H Extra Bones in the Wrist and Ankle, Found by Roentgen Rays, Am J Roentgenol 8 569, Oct, 1921
Quiring, D P The Extremities, Philadelphia, 1945, Lea & Febiger
Rao, V R, and Kini, M G Indian Medical Research Memoirs Infections of the Foot, Calcutta, Indian Council of Medical Research, 1933, p 32
Shands, A R, Jr, and Wentz, I J Symposium on Orthopedic Surgery, Congenital Anomalies, Accessory Bones and Osteochondritis in the Feet of 850 Children, S Clin North America 33 1643-1666, 1953
Shephard, Edmund Tarsal Movements, J Bone & Joint Surg 33B 258-263, May, 1951
Trolle, D Accessory Bones of the Human Foot, Copenhagen, 1948, Ejnar Munksgaard Forlag
Turchin, C R Theory, Physiology and Treatment of Foot Imbalance, J Nat A Chiropod 45 17-51, Nov, 1955
Watkins, W W Anomalous Bones of the Wrist and Foot in Relation to Injury, J A M A 108 270, Jan 23, 1937

HISTORY

It is essential to elicit an exact and a detailed history preparatory to careful examination, if the diagnosis is to be correct A careful analysis should be made of the immediate reason that induced the patient to seek relief The onset and course often supply the diagnostic impression from which to proceed toward establishing the diagnosis Close attention to what the patient says and how he says it may prove more indicative than any other means of preliminary search A history of poliomyelitis or other types of paralysis rheumatic fever, or allergies may provide the clue to current disturbances A history of delayed or difficult surgical healing or reaction to anesthesia and drugs will furnish guidance in planning treatment Varicosities are often secondary to multiple pregnancies, pelvic tumors can affect the circulation of the lower extremities Habits may be contributory Buerger's disease (thromboangiitis obliterans) is often found in heavy smokers. Evidence is accumulating that tobacco aggravates vascular deficiencies

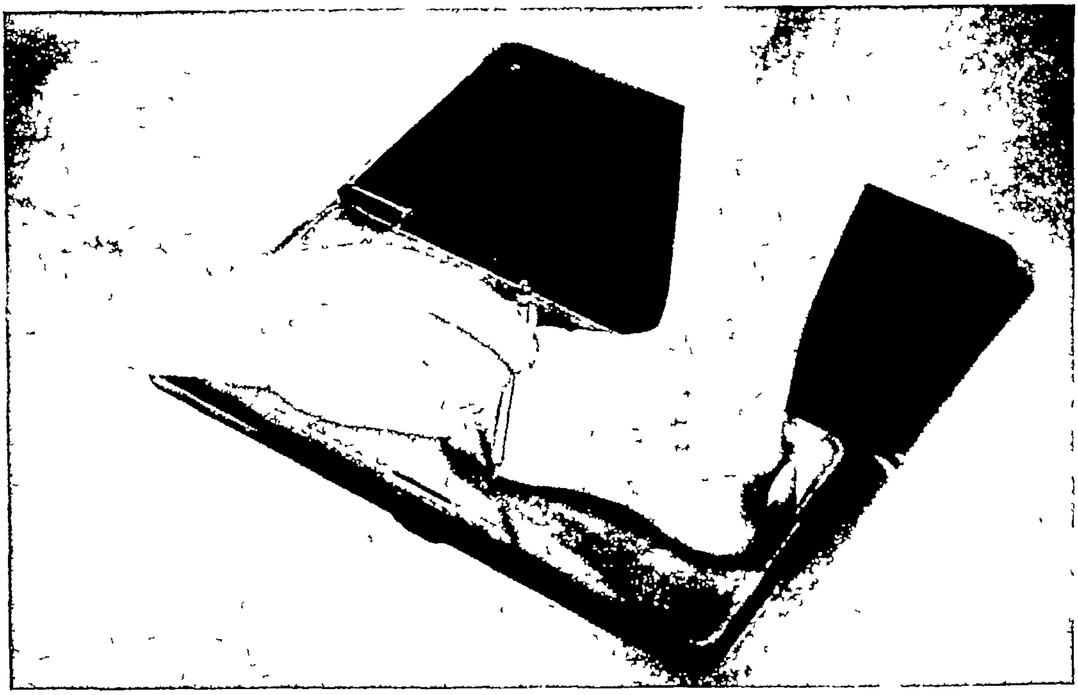
AGE AND SEX

Children are more susceptible to poliomyelitis and to its residual effects on the extremities Separation of epiphyses also occurs early in life Because of vigorous activity, children are more subject to trauma of the foot and to the presence of foreign bodies, such as glass and needles During adolescence and early adult life, acute and chronic effects of ill-fitting shoes become evident Some wear poorly fitted shoes without awareness, others persist in wearing them to be fashionable During middle life the incidence of occupational hazards to the foot rises as does the incidence of varicosities, rheumatoid and gouty arthritis, venereal disease, climacteric changes, thrombophlebitis, phlebothrombosis, and diabetes—all of which have manifestations in the foot Associated with advanced age are arteriosclerosis, cardiorenal diseases, malignancies, ulcers, including trophic, varicose, and congestive ulcers, and degenerative arthritis—each of which often produces early symptoms in the foot

Women are more disposed to wearing faulty shoes, moreover, they experience the additional impairments of circulation in the legs and feet as a result of pregnancy or intrapelvic neoplasms, which are important causes of varicose veins More men exhibit occupational foot disabilities from strain or trauma, of the types seen in policemen, postmen, and laborers Men are more neglectful of foot difficulties than women The incidence of gout is higher in men

GAIT

The patient's gait frequently gives a direct clue to the basic foot problem Gait affected by local disabilities is directly related to the static or acute foot deformities, which the patient reveals by favoring one part over another when walking It is natural to shift to an antalgic gait General diseases may also cause typical gaits ataxic (tabes dorsalis, Friedreich's ataxia), spastic (spinal cord



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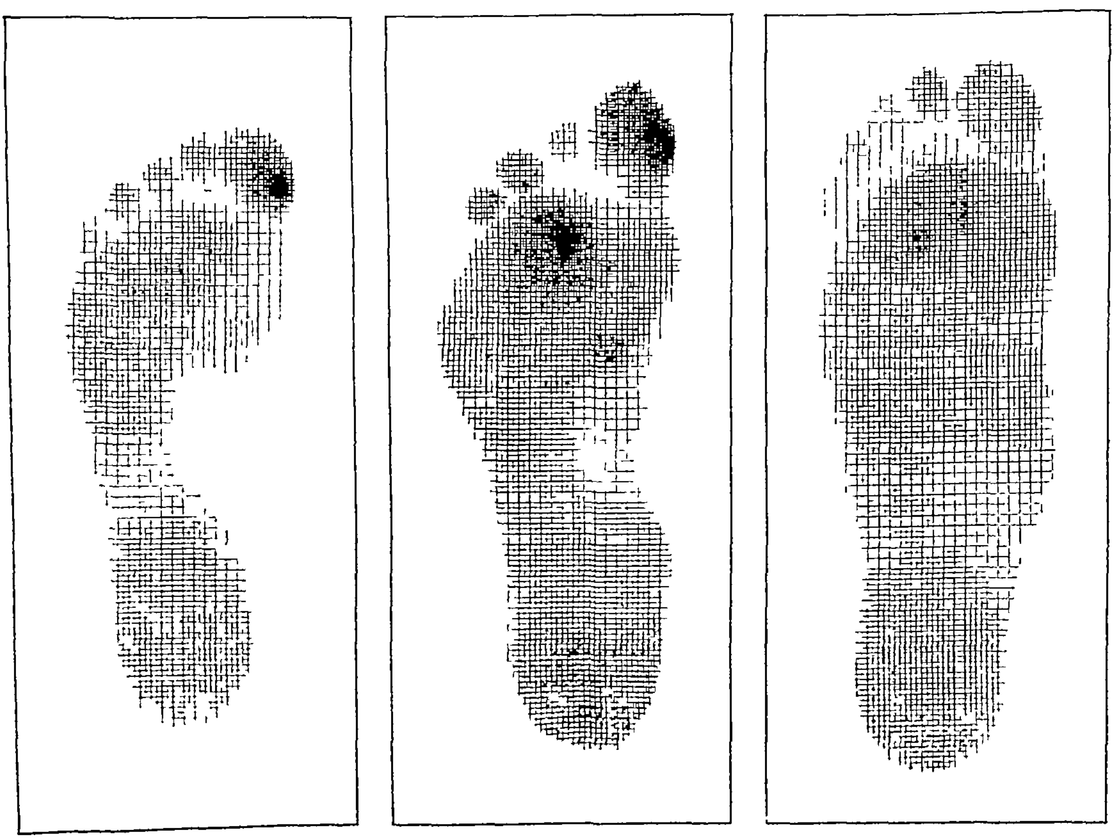


B

Fig 41 —A, Foot being placed on pedograph machine B, Three pedographic impressions Left, Normal Center, Moderate flatfoot, note keratotic lesion under second metatarsal head Right, Third degree flatfoot Note keratotic lesion under third metatarsal head (Courtesy Dr William M Scholl)

Fig 44 —A, Top, Feet in normal stance Center, Normal plantar surface posterior foot in normal position, medial view of normal foot Bottom, Achilles tendon in straight line B, Top, Feet abducted Center, Plantar surface of flatfoot, eversion of foot, medial view of flatfoot Bottom, Foot abducted, Achilles tendon medialward (Courtesy Dr William M Scholl)

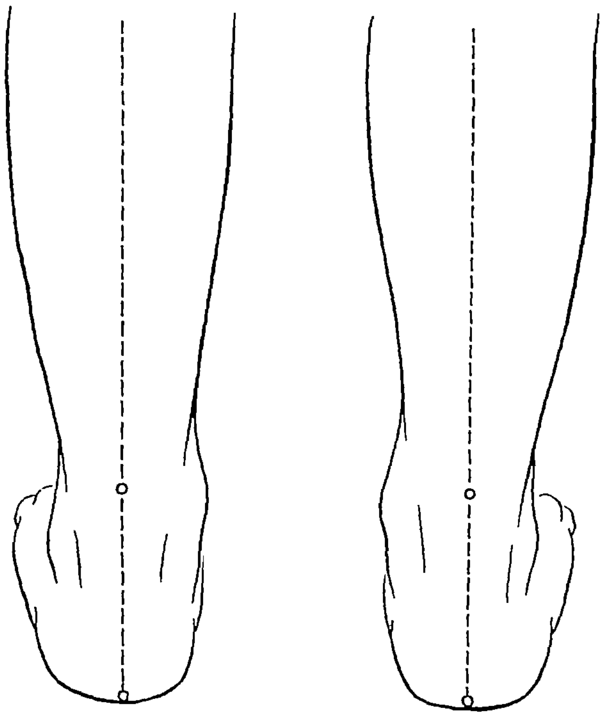


Fig 42 —Achilles tendon in vertical line

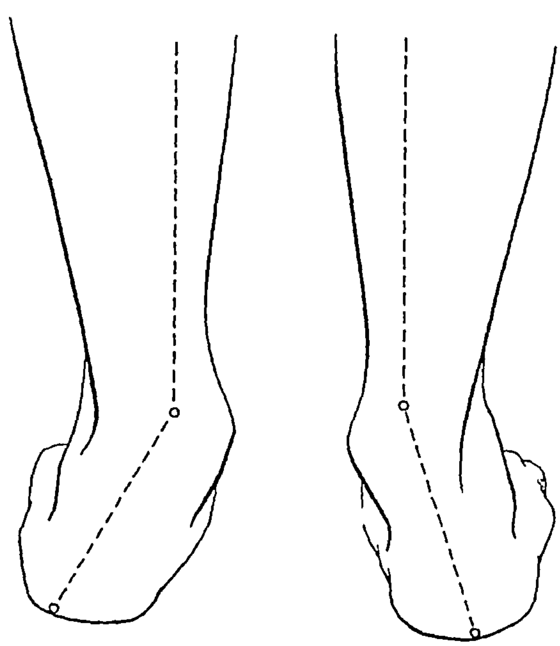


Fig 43 —In an everted foot Achilles tendon is medialward behind medial malleolus

the forepart of the upper part of the shoe, to hold them on the foot, this encourages fitting them short

Orthopedic shoes, equipped with a variety of insoles, arch supports, and other gadgets designed to correct foot disorders, offer no advantages whatever for a child who has normal healthy feet They may even be harmful

The foot would cause man no pain on its own account were it not for systemic disturbances and for modern civilization which disregards the physiology of the foot Fashion, eye appeal, rather than function (Camper, 1792) is for the

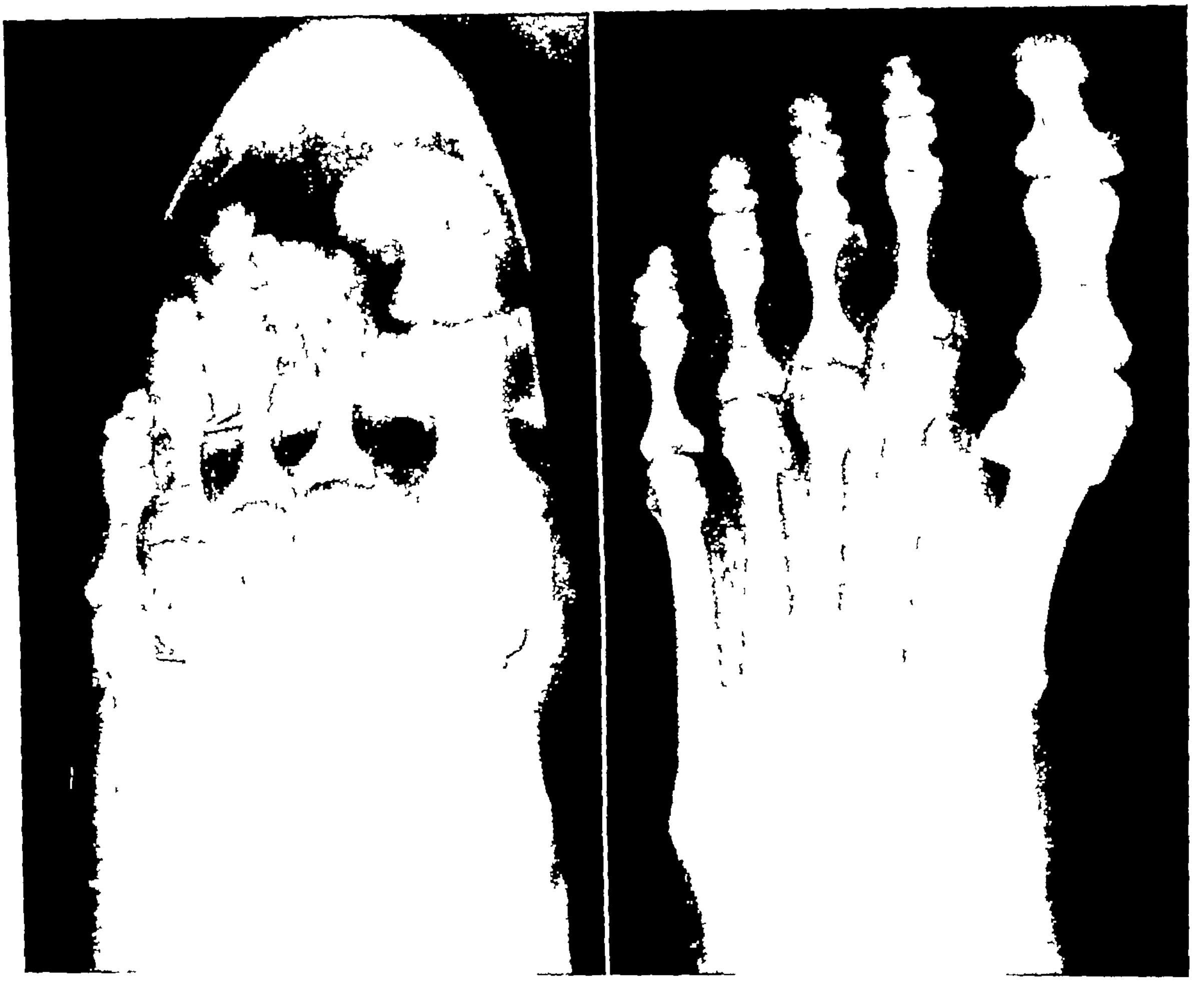


Fig 45—*A*, Foot restricted in pointed shoe notwithstanding apparent sufficient length *B*, Same foot, without restriction Note marker under first metatarsal head deep-seated hyperkeratosis

most part the determining factor in the design of a shoe, especially of its forepart which causes most of the disabilities and deformities of the foot and, in particular, of the metatarsophalangeal region Inasmuch as most of the major muscles of the foot and leg extend into the toes, the important kinetic functions of the foot are centered about the toes The restriction of ill-fitting shoes distorts anatomic parts while inducing malfunction Eighty per cent of all footwear is ill-fitting, especially women's shoes The foot is not constructed to withstand bearing weight in a shoe that is little more than a sole having a strap or vamp

tumors, multiple sclerosis), paralytic (anterior myelitis), scissors (cerebral diplegia), waddling (muscular dystrophies), and steppage (peripheral neuritis)

The manner in which the foot is used should be observed as the patient walks about without shoes at his customary gait The behavior of the foot should be observed during locomotion in order to ascertain whether the arch is of normal height or depressed and to ascertain the direction in which the toes point forward, outward, or inward So-called normal pointing is not the same for everyone Clemmesen (1957) says "The deviations of the axes of hip, knee and ankle joints cause bound rotations of hip and ankle joints during gait, while the axis of the knee is kept in the frontal plane . "* (The axes and the angles between them vary from person to person and even between races) It is false to regard " a certain foot angle as the normal one Whether an individual should toe in or toe out should be determined essentially by the relationship between the axes "*

The position of the Achilles tendon should be noted Normally, it should be in a vertical line, with its insertion into the posterior tuberosity of the calcaneus (Fig 42) and immediately behind the tibia-talar articulation In an *inverted* foot, the tendo achillis is lateralward, nearer the outer malleolus, whereas in an *everted* foot, it is medialward, behind the medial malleolus (Figs 43 and 44)

SHOES

The infant foot does not require a shoe, although soft loosely fitting shoes may be worn without harm The foot develops better without shoes, however, shoes are necessary for protection while learning to walk on hard surfaces The shoe should have a sole and heel thick enough for that protection but pliable enough to permit normal function It is important that the shoe does not interfere with or restrict the development and normal use of the foot The toes especially should have complete freedom

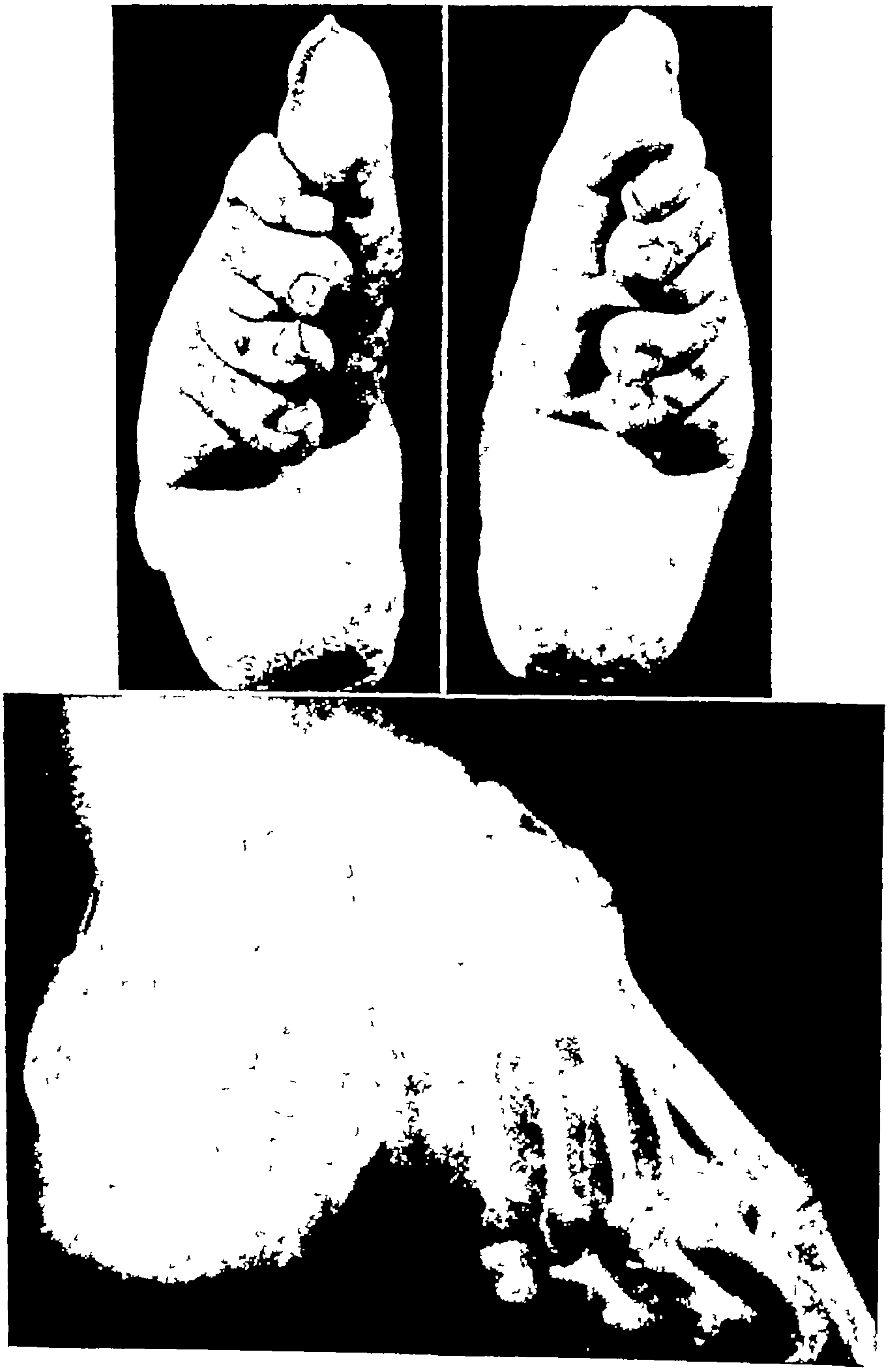
Nature never intended the foot to be shod Comfort is only a partial guide to proper fitting It is essential that a shoe allow free movement of the muscles and joints of the foot and not restrict circulation Short shoes, in the sense that the forepart of the shoe restricts the freedom of the digits, especially if worn during childhood, are the commonest direct cause of foot deformities and lesions

Children, especially girls, commonly outgrow the length of shoes before they are worn out, this should be watched carefully or irreparable injury may result

Warmth and ventilation are to be considered in the selection of a child's shoe, such as a well-designed high shoe in winter and sandals for summer Loafers should be discouraged because the wearer depends upon the vamp, which is

*From Clemmesen, S. M The Influence of Shoes on Deportment and Gait, Postgrad Med 21.43, Jan , 1957

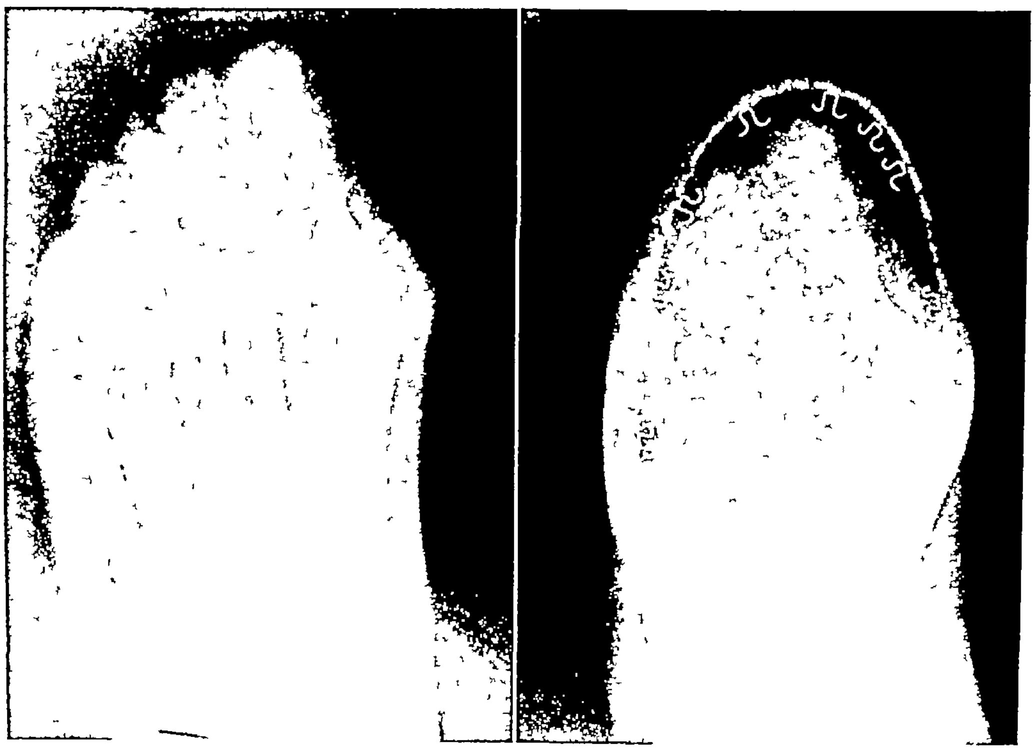
A



B

Fig 47 —*A*, Medial view of bound foot of a Chinese *B*, Roentgenogram of same foot (From Miltner, L J J Bone & Joint Surg n s 35 314-319, April, 1937)

to hold it on the foot and a 3 or 4 inch heel, the base of which is 2 cm in diameter Short or pointed-toed shoes (Fig 45) rather than narrow shoes, in the sense that its circumference at the ball of the shoe is less than the circumference of the metatarsal heads, cause most ordinary foot ailments That is because all blood vessels, nerves, and most of the muscles of the foot course in a longitudinal direction A shoe tight enough to cause deformity would act as a tourniquet, the pain of constriction would be unbearable A short pointed-toed shoe buckles the



A B

Fig 46 —A, Hallux valgus, without shoe B, Same foot in what may be considered a properly shaped shoe, nevertheless, severe restriction of toes accentuates deformity

joints of the toes and metatarsals, but, although it is uncomfortable, it is tolerable, however, over a long period, deformities and excrescences are produced (Fig 46) The Chinese custom of deforming children's feet was accomplished by binding the feet in the longitudinal axis (Fig 47) Prolonged longitudinal restraint produces static deformity

Because of the foregoing explanations, the pattern of wear on the sole and heel of a shoe informs the examiner Any discomfort experienced during weight bearing forces favoring of the affected area, thus, too much exertion on another part of the foot is reflected in excess wear on the sole or heel of the shoe A well-fitting shoe worn on a normal foot shows even wear over the entire forepart of the sole and heel, whereas a shoe worn on an abducted foot shows excess wear on the inner border of the shoe as well as a tendency to flattening of the shank A short shoe shows maximum wear farther forward on the sole The inside of a shoe reveals relative fit of the shoe

flammation. Reactive enlargement is an insidious result of chronic friction and irritation. Neoplastic enlargement is due to new insidious growth of cellular tissue. Edema and induration are always due to some form of stasis of fluids. Inflammation may be due to trauma, infection, or an irritating agent, or it may occur as secondary to a new growth or to edema.

Pressure on Bony Prominences.—Most of the minor and many of the major foot disabilities and morbidities are basically related to friction, pressure, and resistance to which the bony prominences of the foot are subjected. The foot is encased in leather or comparable nonresilient material which seldom follows the contour of the foot and is made to carry the body weight on surfaces that Nature did not intend. Prominences of bone become fulcrums, soft tissue structures covering the prominences undergo morbid changes from prolonged trauma.

A prominence of bone under a keratotic lesion of the foot is often spoken of as an *exostosis* which is a pathologic entity; actually, prominences of bone are normal condylar surfaces. Normal condyles vary in size and shape, hence, the variation in their morbid changes. A low-grade chronic inflammatory process sets up as a reaction to persistent trauma over long duration. At the same time the skin accumulates many layers of horny cells for protection, and the subcutaneous tissue becomes infiltrated by connective tissue. The entire thickness of the horny layers become depressed into the soft structures while the original pressure and friction continue; thus, the horny layers add to the injury of the soft tissues underlying them.

EXAMINATION OF LOWER EXTREMITY

The nails, skin, pulse, color, veins, gait, reflexes—all should be observed during examination of the foot and leg.

Nails.—Compare the toenails with the fingernails, making note of any deformity in either, as well as any brittleness, ridges, or yellow powdery substance under the nail. Brittleness may indicate hypothyroidism, ridges may indicate deficiency states, yellow powdery substance under the nail means the presence of *trichophytosis*. (See Chapter 9, Diseases and Deformities of the Toenails.)

Skin.—Texture and pigmentation alterations are symptomatic of advanced diabetes or vascular diseases. The texture of the skin becomes puttylike. Dark pigmentations appear in the skin surrounding chronic ulcerations and varicosities.

Temperature.—Temperature is suggestive of advanced or terminal vascular disease in which the affected extremity will be colder than the other extremity. The examiner need only place his hands on the feet or legs to ascertain the abnormality of temperature. Normally the temperature of the two feet is the same and is 10 degrees lower than the rest of the body. When unilateral vascular deficiency is present, the temperature is lower in the affected foot.

Pulse.—Vascular deficiences may be differentiated by strong, weak, compressible, or absent pulses. The pulses of the dorsalis pedis and posterior tibial artery are important diagnostic signs in foot disabilities. Absence may indicate obliterative endarteritis, arteriosclerosis (Fig. 48), or functional arterial spasm. The dorsalis pedis cannot be elicited in some normal persons. In advanced arterio-

CRITERIA FOR PROPER FIT OF SHOES

In a properly fitted shoe, the impression on the insole made by the different parts of the foot will be in proper place: the ball of the foot impression immediately anterior to the shank, each toe having its own free impression at least 2 cm from the distal end of the insole. In poorly fitted shoes, the ball impression is forward of the shank and the toe impression is crowded into the far end of the shoe.

Clemmesen (1957) lists criteria* for judging the construction and fit of the appropriate shoe.

Outline of Shoe.—The inward contour of the great toe should continue a line of the inward side of the heel bone. The axis of the heel bone should be vertical. The tip of the shoe should follow the outline of the loaded foot with stocking on; the inner part of the tip of the shoe should be higher, as the great toe is thicker than the other toes.

Length.—The length of the shoe should be tested for both the weight-bearing and non-weight-bearing foot. Use does not lengthen a shoe.

Width.—The width of the shoe should be mildly snug around the metatarsal heads during standing and walking.

Size.—There must be room to allow the toes to bend, stretch, and part during gait. Those who stand and walk a great deal need larger shoes than others.

Sole.—The sole must be broad enough to prevent the side of the foot from sagging outside the edge of the shoe. This happens if the side of the foot must press against the shoe. The sole must be almost level from side to side.

Heel.—Heel piece and counter should fit closely. Height and width of heel must be controlled. A shallow bowl-formed surface, higher on the inner than outer side, gives good heel support. A Thomas heel also gives support. The heel support must prevent the foot from slipping into the shoe.

Counter.—The counter should fit closely. It should not reach the tuberosity of the fifth metatarsal bone.

Shank: Toe-Off Phase of Gait.—The great toe must not be forced toward the middle of the foot when the foot slides forward in the shoe during the toe-off phase of the gait. Shank should be hard enough to support the longitudinal arch in front of the heel, but it must not be so stiff as to prevent the break of the sole in the toe-off phase of the gait.

Girth and Upper Edge of Shoe.—The girth must not sag; the ankle bones must not touch the upper edge of the shoe during movements of the foot.

TOPICAL OBSERVATIONS

When evaluating a disorder of the foot in which systemic symptoms are present, it is necessary to determine at the outset whether the disease is a local process in which there is mild systemic reaction or a systemic disease having local symptoms. The general appearance of the entire foot must be appraised by comparison of the two feet, by observing the relation of the foot to the ankle and leg, by eliciting points of tenderness to pressure, and by checking for deformities of the forefoot. Adduction or abduction indicates *intrinsic* deformity in the tarsometatarsal joints or *extrinsic* deformity in the ankle, knee, or hip joints.

Hypertrophies and Hyperplasias.—Any increase in size of a tissue other than that occurring in normal growth represents one of the following: (1) neoplastic enlargement, (2) reactive enlargement, (3) edema and induration, or (4) in-

*Clemmesen's twenty criteria are submitted here without enumeration and in altered arrangement for condensation. Although the paraphrase does at times follow the wording, none of the points is quoted fully. The original article is recommended for detailed reading.

DIFFERENTIAL DIAGNOSIS

Most foot morbidities fall into one of the following groups

1 Common excrescences, neoplasms, pressure on a condylar surface, static deformities, structural malfunction In this group, the symptom can usually be pinpointed to make the diagnosis. Location of the affected area on the roentgenogram and on a skeleton of the foot facilitates the diagnosis.

2. Conditions due to vascular deficiencies, such as venous stasis secondary to varicose veins or the arterial diseases The symptoms in this group are vague and generalized The area of pain cannot be pinpointed. In the later stages of arterial disease, intermittent claudication, pain, blanching, ulceration, and paresthesia appear in the foot proper. Decisive and rapid color changes take place on elevation and dependency The oscillometric index will be diminished or absent Edwards (1956) has described a syndrome which he calls *remittent necrotizing acrocyanosis* in which there is symmetrical coldness, pain, and cyanosis in hands and feet. "Functional vascular spasm and organic occlusion are both factors in this disease . . Perivascular inflammation, intimal proliferation in the arterioles of the skin, and occlusion of the capillaries of the skin by hyaline thrombi were characteristic findings "* Apparently that syndrome is not related to Buerger's or Raynaud's diseases and in some of his cases required amputation of digits

3 Morbidities due to trophic changes, such as injury or irritation to the lumbar sacral nerve root Pain may be vague, more so than in vascular disease The patient may say that the foot feels peculiar. Symptoms of tingling and numbness are often referred to the plantar surface The pulse and oscillometric reading are normal Except in terminal cases there are seldom any changes in the tissues of the foot Reflexes may or may not be altered Investigation of the lumbar sacral architecture may disclose the source of the problem

4 Morbidities due to general diseases Symptoms are variable and may or may not be pinpointed The diabetic patient often has ulcerated areas over the toes or under the ball of the foot, usually on the condylar surfaces, and invariably some vascular deficiency of the foot is present Gout often produces changes in the metatarsophalangeal joints, especially of the first joint

INTERPRETATION OF PAIN

Pain in the foot is the single important symptom from which the patient seeks relief no matter what morbid conditions are present For that reason, the patient's description of the pain is likely to be the most important clue to diagnosis

The bones of the body are designed in conformity with their function. For that reason, long periods of standing or walking in shoes constructed without regard for the structure and function of bones result in unrelenting pressure and irritation over normal prominences of bone and cause those common pains in the foot which are not caused by disease

*From Edwards, E A Intermittent Necrotizing Acrocyanosis, J A M A 161 1530-1534, Aug 18, 1956

sclerosis, there is evidence in the roentgenogram of calcification of the arteries "Occlusion of main vessels can be identified by the simple expedient of feeling the pulses No operation on the foot should be undertaken without due consideration for its blood supply "*

Color.–Observe the color when the leg is dependent and when it is elevated, how long the color takes to change from dark to a light pink Normally, this takes about five minutes When the color is white on elevation and beet red immediately on lowering, it is an indication of terminal vascular disease

Fig 48 –*Arteriosclerosis of dorsalis pedis*

A wealth of information may be had from the intelligent use of the clinical laboratory Routine laboratory analyses prevent embarrassments of unawareness on the surgeon's part, but what is more important, they prevent needless complications for the patient It is, however, as much of a fault to depend entirely on the laboratory for a diagnosis as it is to neglect its use in making a diagnosis Urinalysis, complete blood counts, and serologic tests are prerequisites to surgical intervention in the foot When laboratory reports leave the patient's status equivocal, further investigation must be pursued before a surgical procedure is undertaken.

*From Henson, G P A Case of Unsuspected Arterial Disease, Brit J Surg 43 665-666, May, 1956

the foot must flow farther against gravity than from any other part of the body Edema is symptomatic of general disorders that produce a stasis of fluid in soft tissue It means that normal interchange of fluid has been disturbed Systemic diseases that cause accumulation of fluids are renal disease, cardiac insufficiency, hypothyroidism, diabetes mellitus, general debility, allergy, severe anemia from whatever cause, Milroy's disease, obesity, pelvic growths, pregnancy, endocrinopathies, angioneurotic edema, and avitaminosis Local conditions causing edema may be occupational, traumatic, such as from sprains or small ununited chip fractures, local arterial deficiency, weak foot; Sudeck's atrophy, or may represent a climatic reaction to excessive humidity and heat

Edema of the foot and ankle caused by local conditions are usually unilateral and nonpitting, whereas general conditions usually cause bilateral pitting edema Cardiac insufficiency is usually a cause of an *inconstant edema* which disappears when in a lying down position, whereas the other general diseases cause *constant edema* in which only slight recessions take place

REFERENCES

Camper, P Dissertation sur les variétes naturelles qui caracterissent la physionomie des hommes des divers climats et des différents ages, dissertation, sur la meilleure forme des souliers, Paris, 1792, Francart

Clemmesen, S M The Influence of Shoes on Deportment and Gait, Postgrad Med 21 43, Jan , 1957

Edwards, E A Intermittent Necrotizing Acrocyanosis, J A M A 161 1530-1534, Aug 18, 1956

Fairbank, H A T From Atlas of General Affections of Skeleton, Paget's Disease, Synonym—Osteitis Defomans, J Bone & Joint Surg 32B 253-265, May, 1950

Henson, G P A Case of Unsuspected Arterial Disease, Brit J Surg 43 665-666, May, 1956

Lassèrre, C , and Clavé Maladie de Paget révélée par une talalgie, J de méd de Bordeaux 117 245-247, April 20-27, 1940

Murray, J G Tender Heel Due to Paget's Disease, J Bone & Joint Surg 34B 440-441, Aug , 1952

Pain in Specific Areas of Foot.—Pain in the foot can often be pinpointed at specific areas for differential diagnosis

Great Toe—Stresses applicable to the great toe are not applicable to the lesser toes It will be recalled that the great toe has only two phalanges and has four intrinsic muscles inserted into its proximal phalanx The phalanges of the great toe are comparatively large bones and absorb more shock in each step than do all the lesser toes together At times the great toe assumes the entire weight of the body as in the case of a toe dancer. As a result, pain-producing changes often arise in that toe Gout also commonly involves the great toe and is characterized by acute recurrent attacks of severe pain and swelling

Great Toe Joint—Functionally the most important and most complex joint in the foot is the great toe joint Bursitis, hallux rigidus, hallux valgus, ganglionic cyst, displaced sesamoid, sinus into joint cavity, and hypermobile first metatarsal as seen in metatarsus primus varus—all cause pain and all except the hypermobile first metatarsal are due to poorly fitting shoes, especially short shoes

Metatarsal Interspaces—Pain in the metatarsal interspaces is often due to neurofibroma (Chapter 13) which commonly occurs in the third interspace but may occur in any other interspace Ganglionic cyst, march fracture, or os intermetatarsum may also produce pain in this area

Longitudinal Arch—Symptomatic pain from a weak foot, neoplasms, infections of tendons or fascial planes, or pain from ill-fitting shoes may be experienced in the longitudinal arch

Under Ball of Foot—Pain under the ball of the foot may be due to Morton's syndrome, verrucae plantaris, epidermal cysts, or dislocation of the metatarsophalangeal joint of the lesser toes

Heel—Pain in the heel may be due to retrocalcaneal bursitis, apophysitis, avulsion fracture, so-called Haglund's exostosis, neoplasms of tendo achillis, infraction of os trigonum, calcaneal spurs, or fibrositis at the origin of the plantar fascia Murray (1952) reported two cases of painful heels due to Paget's disease, the main symptom was of pain in the heel Fairbank (1950) and Lassèrre and Clavé (1940) also reported cases of Paget's disease of the calcaneus with primary symptoms of pain in this bone

Interpretation of Types of Pain—*Sharp* pain may be due to injury, acute infection, pressure, excrescences or tumors *Dull* pain may be due to tuberculosis, syphilis, diabetes, anemia, vascular disease, gout, or to local conditions, such as weak foot, static deformities, or severe strain *Stabbing* pain indicates either the presence of a foreign body or neuritis *Radiating* pain begins at the origin of a nerve or along a nerve's course and radiates distally It is usually due to irritation of a nerve root *Paroxysmal* pain, such as that induced by intermittent claudication, has a sudden onset, as a cramp in the calf of the leg after walking a short distance It is characteristic of vascular deficiencies *Migrating* pain may result from such diseases as rheumatic fever, early stages of rheumatoid arthritis, anemia, or peripheral neuritis

Edema of Foot and Ankle—Edema of the foot may be due to local or general causes The force of gravity often determines the site of edema That is why obviously the foot and ankle are the commonest sites, the return of fluids from

So-called ingrown nails, hammer toes, small tumors, and cysts may be reduced or removed in the office, condylectomy for corns, phalangeal capsulotomy, and tenotomy are procedures safely accomplished in the office.

Assistants

The value of all personnel entering into the operating room depends essentially on their interest and on their ability to act and work as a unit Assistants and nurses should be chosen on the basis of their interest in the field of surgery of the foot.

Preoperative Care

Planning the Operation.—Surgical operations of the foot are usually elective, so that the surgeon has time for thorough evaluation and unequivocal diagnosis Time for planning is an advantage which the surgeon should explain to the restless patient It is well to schedule the date for operation far enough in advance to afford the opportunity of studying the patient and the problem a second or third time before the actual date set Preoperative urinalysis and a complete blood count should be made routinely even for the most minor procedures for the foot. This is an obvious preventive measure.

Instruction to Patient.—Specific instructions to the patient as listed herewith prevent misunderstanding and are helpful to the surgeon (1) shave all hair from foot and leg, (2) scrub the foot, especially around the nails, with soap and brush for ten minutes shortly before leaving for the office or hospital outpatient department, (3) bring along white socks and laced Oxfords which may be cut if necessary to allow for swelling and dressings, (4) do not eat for five hours before the time set for operation

For inpatient hospital procedure, the patient must be instructed to report at the hospital by mid-afternoon on the day before the operation.

The diabetic patient needs special attention but is not denied necessary operation because of the disease It is generally conceded that a controlled diabetic patient, properly prepared, is not a greater surgical risk than is a nondiabetic patient, however, it is safer to consider all diabetic patients as poor surgical risks Even for minor operations, the diabetic patient should be hospitalized where adequate laboratory facilities are had He should be observed carefully by a physician experienced in the treatment of diabetes It is always unwise for the surgeon to treat the diabetic condition.

ARMAMENTARIA

Modern hospitals have special operating rooms and instruments designed for the specialties Surgery of the foot is usually assigned to the orthopedic room which is equipped with the usual orthopedic instruments Because of the small areas involved in operating on the foot, however, most instruments must be small, sharp, and fine, especially for operations on the forefoot For that reason the surroundings and instruments of the rhinologist and otolaryngologist

CHAPTER THREE

Operative Principles and Requirements

PREOPERATIVE CONSIDERATIONS

THE RESULTS OF SURGICAL INTERVENTION DEPEND ON THE COMBINED CARE DURING three phases, preoperative, operative, and postoperative

The surgical process from the incision to the final closure is a succession of steps, each of which exerts a form of trauma Pick (1949) describes the surgical operation as a "premeditated, measured and ingenious form of trauma, with the respectable purpose of ablating certain diseases and bodily defects in man or animal" It is important that every operation be carefully planned, so that the ultimate benefit of the procedure will justify unavoidable surgical trauma It is the iatrogenic, the superfluous trauma, the careless technique, and lack of training which are deplored The process of healing and the final functioning of the tissues are in direct relationship to the degree of operative injury The degree of trauma experienced during a surgical procedure depends on the surgeon's knowledge, judgment, and conscience, on the type of instruments used, the manner of their use, and the handling of tissues, so as to keep to an irreducible minimum the pulling, dissection, tearing, bleeding, sponging, and crushing

Suitable Office Procedures

Surgery of the foot may be divided into problems of the forefoot (metatarsal and phalanges), which comprise 90 per cent of the cases, and problems of the hindfoot (tarsus), which comprise the remainder Half of the procedures for the forefoot and some of the minor procedures for the hindfoot may be performed in the office, provided the office is equipped with an autoclave, appropriate instruments, dressing materials, linens, and all other armamentaria essential to minor surgery Proper preparation and asepsis must be as rigidly controlled in the office as in the hospital

Fig 49 —Author's selection of instruments for foot surgery

1 Eye dressing forceps
2 Graefe fixation forceps with catch
3 5 inch dressing forceps
4 Adson tissue forceps
5 6 inch dressing forceps
6 Tissue forceps, 2 by 3 fine teeth
7 Curved Mayo scissors
8 Straight Mayo scissors
9 Sharp straight scissors (both points sharp)
10 Strabismus straight scissors
11 Sistrunk scissors, DuVries' modification
12 Littauer suture scissors
13 Spencer suture scissors
14 Backhaus towel clamp
15 Curved mosquito forceps
16 Kelly straight forceps
17 Straight mosquito forceps
18 Scalpel blade, No 10
19 Scalpel blade, No 11
20 Scalpel blade, No 15
21 Mayo-Hegar needle holder
22 Collier needle holder
23 Fine cat's-paw retractor
24 Cat's-paw retractor with flexible neck
25 Allis forceps
26 Allis forceps with jaws curved at right angle
27 Bunnell tendon guide, long
28 Bunnell tendon guide, short
29 Flexible wire threader
30 Small curette
31 Nasal saw with short neck.
32 Maltz rasp
33 Joseph nasal rasp
34 Sayre periosteal elevator
35 Mallet
36 Andrews mastoid osteotome
37 8 mm osteotome, Stille pattern
38 10 mm osteotome, Stille pattern
39 7½ inch double-action bone forceps, DuVries' modification
40 Hibbs bone-cutting forceps, DuVries' modification
41 Weitlander self-retaining retractor
42 Self-retaining wound retractor
43 Keith needle
44 Anchor brand 1822, No 16 cutting needle
45 Anchor brand 1822, No 18 cutting needle
46 Anchor brand 1832, No 8 cutting needle

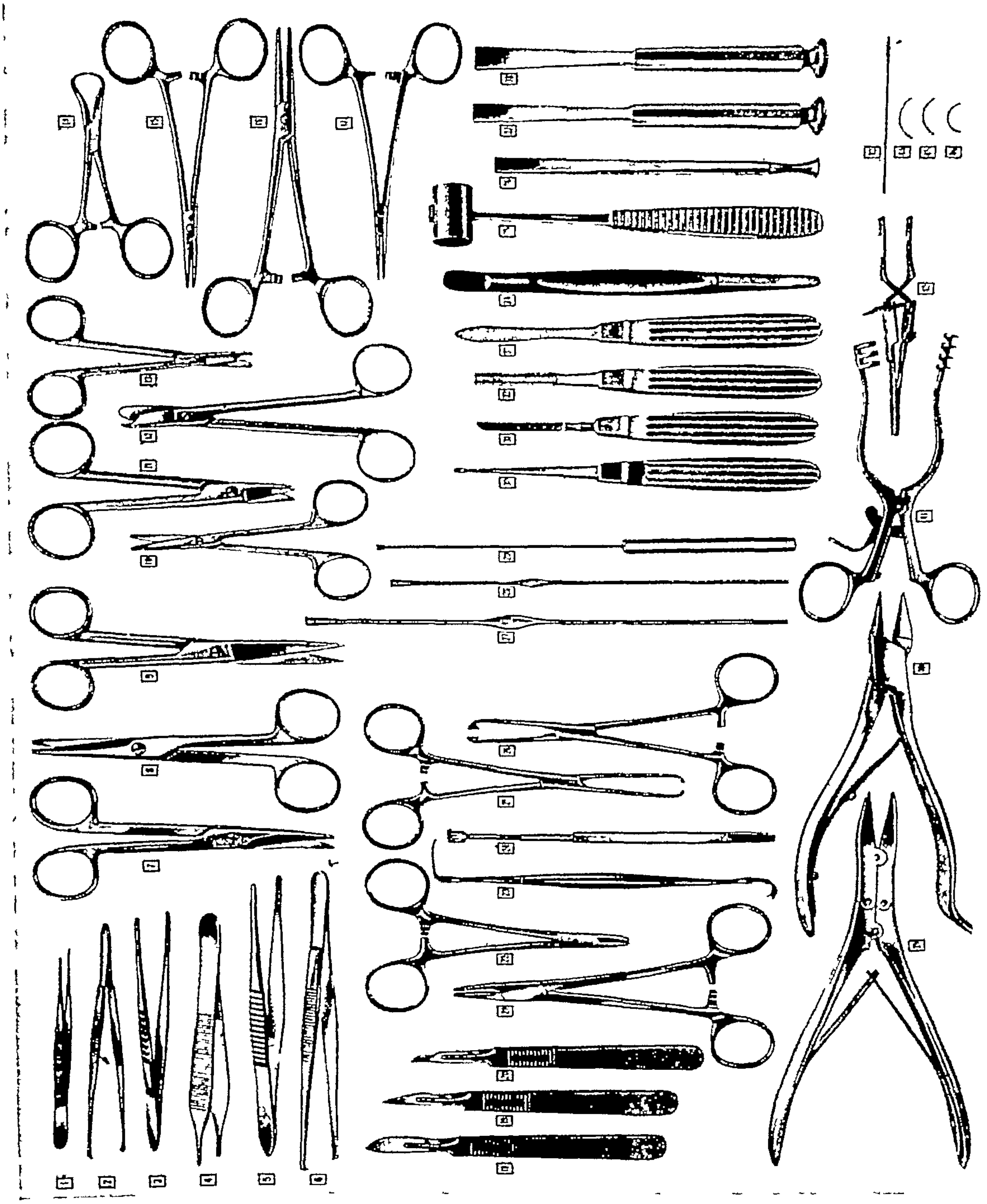


Fig 49—For legend see opposite page

foot, (3) sterile gauze sponges, (4) Telfa squares, to be applied over incisions or raw surfaces, so as to prevent the dressing from adhering to the wound, (5) sponge-rubber sheets, ¼ and ½ inch for padding, (6) ⅜ inch piano felt for padding, (7) paraffined lace mesh for draining ulcers, (8) lamb's wool for protection between the toes.

Syringes and Hypodermic Needles.—For general hypodermic injections, such as for local anesthesia, the 4 ml Luer-tip syringes are handy. Hypodermic needles need not be any heavier than 24 or 25 gauge and no longer than 1 inch.

Tourniquets.—The main types of tourniquets are the Martin sheet rubber bandage and the Campbell-Body, Conn, or Kidde automatic tourniquet (Fig. 50). Tourniquets over the leg or thigh should always be applied over towels to prevent injury to the skin. For most digital surgery, a common rubber band at the base of the toe is sufficient to leave the field bloodless for the short duration required in most instances.

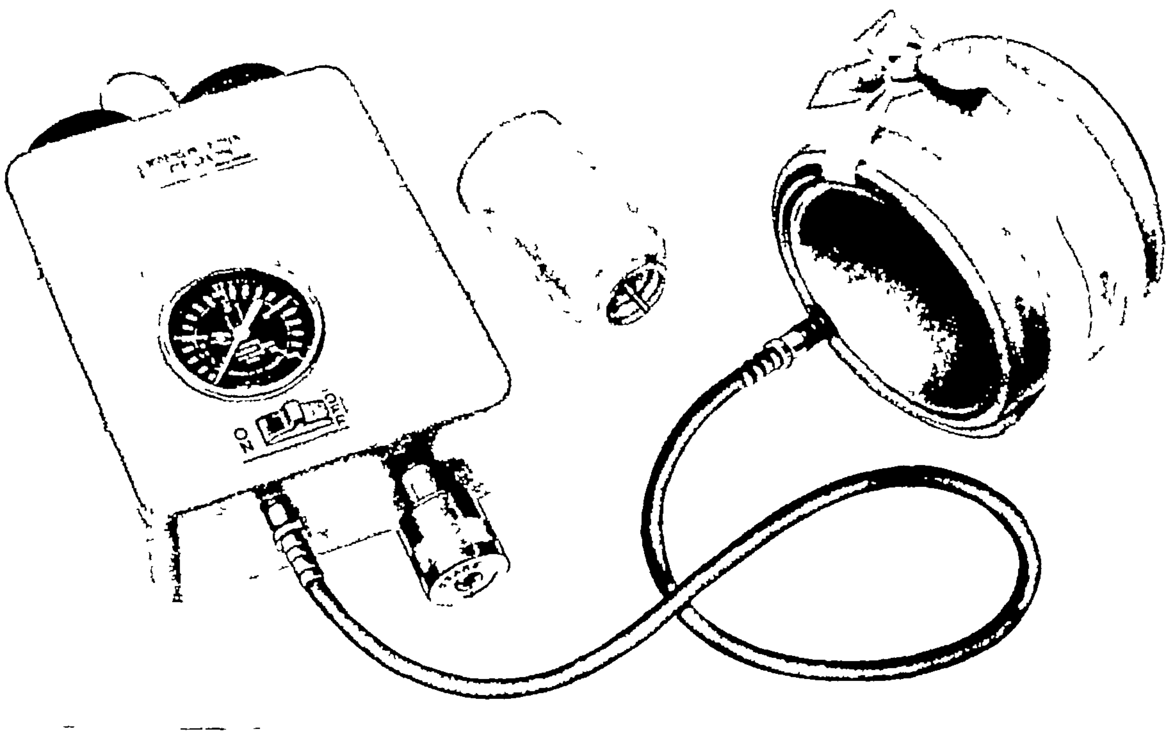


Fig. 50.—Kidde automatic pneumatic tourniquet. (Courtesy Kidde Manufacturing Co., Inc., Bloomfield, N.J.)

OPERATIVE CONSIDERATIONS

Hemostasis

Most foot operations are performed best under hemostasis, which obviates the need for ligatures. Immediate postoperative bleeding is controlled by the application of a compression bandage before release of the tourniquet, which must be loosened between twelve and twenty-four hours.

Although the use of tourniquets is not without danger, their judicious use in surgery of the foot is helpful because of the small structures that must be

are more favorable than the orthopedic room. The foot surgeon must see that suitable armamentaria are provided by the hospital or else he must supply his own For major surgical procedures of the ankle, general orthopedic instruments are utilized

Essential Instruments.—The following instruments (Fig 49) are essential for most surgical procedures on the forefoot, whether performed in the office or in the hospital two Bard-Parker handles, No 3, six No 10 blades, six No 11 blades, six mosquito (Andrews) forceps, curved, six mosquito (Andrews) forceps, straight, two pairs tissue forceps without teeth; two pairs Allis forceps, one Lane periosteal elevator, four Backhaus towel clamps; two fine cat's-paw retractors, two Hegar or Mayo needle holders, 6 or 7 inches long, two straight nasal saws, one pair DuVries bone-cutting forceps (in which jaws come to a point), two pairs curved Sistrunk scissors (standard Sistrunk is too heavy for foot surgery, therefore, an instrument maker can cut down the standard 6 inch curved Mayo scissors to the Sistrunk shape), one pair Stevens straight scissors, one pair Mayo 6 inch straight scissors, one Jameson mastoid retractor or Weitlander retractor, two Joseph nasal rasps, one fine, one medium, one Andrews mastoid osteotome, one pair Littauer suture scissors, one mallet, one 8 mm osteotome This list is by no means complete but represents basic needs, other instruments are employed as required

Suturing Needles—Just as the instruments for most procedures on the foot must be small, so must the needles be of small caliber for use on small areas and minute tissues A large needle becomes unmanageable and traumatizing Because tendons and fascia are so frequently involved, to forestall breakage, the needle should have a cutting edge and be thicker than those small needles used on membranous tissue A broken needle in a deep wound in the foot can be difficult to locate The following needles fulfill most of the needs in foot surgery six No 1848, Anchor brand, size 1 for skin, six No 1848, Anchor brand, size 2 for skin, six No 1834, Anchor brand, size 8 for fascia, six No 1834, Anchor brand, size 9 for fascia

Sutures.—A suture is primarily a splint or ligature, as a splint, its function is to hold tissues in coaptation until union has taken place As a ligature, its function is to act as a pursestring around a vessel, so as to control hemorrhage, or around a structure that is to be divided Because all sutures are foreign material, they are irritants, it is therefore essential to select a suture or ligature that will fulfill a particular need with the least amount of irritation for routine surgical procedures, fine catgut, plain or chromic, Nos 2-0 to 4-0 for tendon sheath and capsule repair, and fine silk or wire for suturing fascia, tendon, and skin Dettinger and Bowers (1957) conducted a comparative study of Orlon, Dacron, monofilament nylon, cotton, and silk sutures and concluded that the synthetic fibers were less irritating to tissues, Dacron least of all They recommend further clinical evaluation

Dressing Materials—The following dressing materials should be at hand in an office or hospital surgical dressing room (1) a 1 inch gauze roller for bandaging of the toes, (2) a 1½ inch gauze roller for bandaging of the fore-

Local (Regional) Anesthesia*.—Most minor procedures on the foot can and should be done under local anesthesia administered by the surgeon. The successful use of a local anesthetic depends as much on the technique of administration as on the inherent property of the agent. Methods of inducing local anesthesia are by application of cold to tissues, by pressure on a nerve trunk, by chemical irritants to the skin which injure or kill nerve endings, such as phenol, and by drugs that can be injected or applied topically to membranes, inducing anesthesia without destroying the tissues as chemical irritants do.

Ischemia has a local anesthetizing effect. Drugs are the safest, most reliable, and most practical agents, because they are relatively noninjurious to nerve fibers and cells and because their action is completely reversible. Idiosyncrasies to drugs, however, are not uncommon. When such idiosyncrasies seem likely, a skin test should be made by the patch method or by intradermal injection.

Local anesthetics are administered by the following procedures: topical application, in which the drug is applied or sprayed on a surface of the body, intradermally, in which the anesthetic is injected into the skin, subcutaneously, in which the anesthetic is injected into the field of operation, and by nerve block or block anesthesia, in which the anesthetic is injected around a nerve trunk, proximally to the area of operation. A local anesthetic when applied to nerve structures blocks the transmission of the peripheral nerve impulse. All local anesthetics may cause stimulation or depression of the sensory and motor cortex, as well as of other parts of the central nervous system. Overdosage may cause convulsions and other reactions.

Procaine hydrochloride is the most popular and probably the safest of all local anesthetic agents because it is the least toxic and is destroyed rapidly in the body, it is nonirritating to tissues in 2 per cent solution, and half the original dose can be repeated in twenty minutes without causing toxicity. It is in standard use for infiltration anesthetics. For extensive infiltration anesthesia, procaine hydrochloride should be used in a concentration of 0.5 per cent with epinephrine 1:200,000 as the local vasoconstrictor. For small areas a concentration of 2 per cent with 1:50,000 to 1:200,000 epinephrine may be used.

Substitutes for procaine hydrochloride in infiltration anesthesia are β-diethylaminoethyl-*p*-ethoxybenzoate hydrochloride (Intracaine), piperocaine hydrochloride (Metycaine), lidocaine hydrochloride (Xylocaine), and butethamine formate (Monocaine). Because cocaine is unstable, toxic, and narcotic, it has no place in surgery of the foot.

Precautions in Infiltration Anesthesia.—All local anesthetic agents are potentially toxic. The safe dosage of each drug is limited by the tolerance of the patient. The following factors determine safe dosage: total amount used, rate of injection, type of premedication, age of patient, emotional and physical status.

General and Special Precautions.—Question the patient regarding known sensitivity to the drug or class of drugs to be administered, autoclave ampules when possible, otherwise store in alcohol. Authenticate labels of all solutions

*In conformity with recommendations of A. M. A. Fundamentals of Anesthesia, ed. 3, Philadelphia, 1954, W. B. Saunders Co.

handled. Bunnell (1951) explained the purpose when he said, "You cannot repair a watch in an inkwell." A bloodless field minimizes the possibility of infection and prevents trauma by disposing of sponging. A clearly visible anatomic field reduces handling. The following procedure for hemostasis is effective for most surgical procedures of the forefoot.

1. Wrap the scrubbed foot and leg in sterile towels.
2. Apply a Martin bandage tightly in layers over the foot, beginning at the toes and ending just above the ankle. This will effect hemostasis in the foot and ankle.
3. Apply another Martin bandage, beginning just above the end piece of the first bandage and continue it snugly to about midcalf, then fasten.
4. Remove the first bandage from over the foot and ankle.

Complete hemostasis of the foot is safe for about an hour. In bilateral cases, it is best to sterilize the rubber bandages, whereas in unilateral cases, the two-pair sterile glove technique may be employed—the operator's assistant, after he has scrubbed, put on sterile gloves and applied nonsterile bandages, then removes the gloves and puts on sterile gown and gloves.

For extensive surgery of the foot or ankle, a pneumatic cuff at midthigh is most efficient. The cuff is applied over a towel while the patient is in the preparation room. Just before making the initial incision, the leg is elevated 45 degrees for three minutes. The tourniquet is inflated before lowering the limb in order to prevent filling of the superficial veins before the arterial blood flow has been occluded. The pressure required on the average thigh is about 600 mm Hg or about 12 pounds. Children and slender adults require less pressure, whereas persons having large muscular thighs may require higher pressure. In the average healthy adult under 60 years of age, it is safe to leave a tourniquet-inflated midthigh for 1 to 1½ hours.

The tourniquet should not be applied until the patient is well anesthetized, for muscle spasm may permit blood to pass under the tourniquet into the arteries after the muscles have relaxed; furthermore, it will also reduce the time during which the tourniquet is active. Improper use of tourniquets may result in impairment of the blood supply or injury to the nerve immediately under constriction. Tourniquet paralysis may result from excessive pressure or from leaving the instrument on too long. Application of a tourniquet over an area where a nerve trunk courses superficially, as does the common peroneal nerve at the neck of the fibula, may produce permanent injury such as paralysis. Pressure of the tourniquet over the popliteal space may produce thrombosis of the popliteal artery. Insufficient pressure may result in a passive venous congestion and consequent tissue impairment.

Anesthesia

Most major operations on the foot need to be done under general or spinal anesthesia.

Areas for Surgical Approach.—The following areas are satisfactory for surgical approach because scar tissue in these areas is least likely to become a point of irritation: (1) on the dorsum of the forefoot, the areas over the metatarsal interspaces, (2) on the dorsum of the toes, a transverse or longitudinal incision on either border, (3) on the dorsum of the tarsus, an incision along the border of the major tendons, (4) the plantar surface, approached through an incision anywhere along the borders of the plantar skin or the nonweight-bearing surface of the longitudinal arch, (5) for an approach to the plantar surface of the metatarsal heads, an elliptical incision just proximal to the flexor crease of the toes, (6) the great toe joint, approached on the dorsum through an incision over the first metatarsal interspace, on the plantar surface, exposed by an incision along its medioplantar border

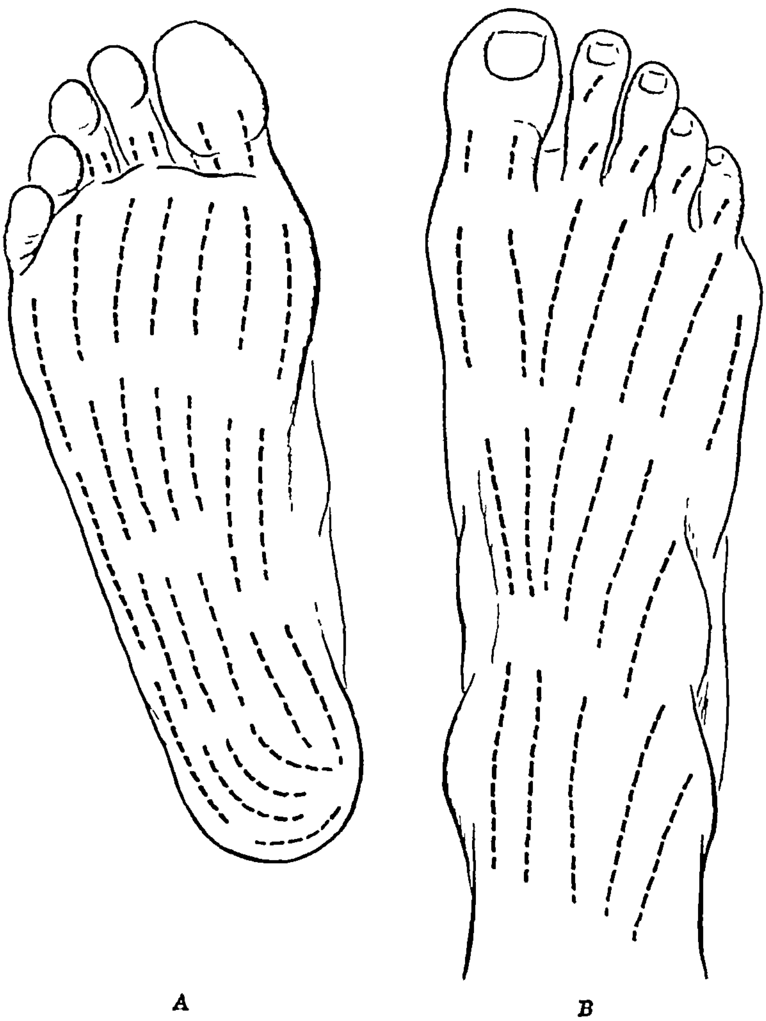


Fig 51 —Skin cleavage *A*, Plantar surface *B*, Dorsal surface

Suturing.—The surgeon must train his fingers by persistent practice of well-selected exercises, as outlined by Tauber (1955) and Partipilo (1957), if he is to become adept at suturing a wound properly, that is, with the least traumatizing and with the greatest methodical care and skill in handling small units of tissue

before administration of drug Do not inject into an inflamed area Such injections are painful, ineffective, spread infection, and increase toxic absorption.

Vasoconstrictors—A vasoconstrictor, such as epinephrine, added to a local anesthetic decreases the vascularity of the operative field and diminishes the rate of absorption of the local anesthetic agent into the system It also prolongs the local action of the anesthetic The average amount of epinephrine used in solutions of a local anesthetic is 1 minim of 1 1,000 solution in 10 ml of 1 per cent solution of procaine hydrochloride

Local Anesthesia of the Foot; Subcutaneous Infiltration.—Injection of an excessive amount of the anesthetizing drug or its introduction under severe pressure or both abuses may destroy the local capillary beds, resulting in extreme postoperative pain and possible sloughing of the part

Most forefoot conditions responsive to local anesthesia involve small areas, such as the toes or the metatarsophalangeal region, which rarely require more than 2 or 3 ml of the anesthetic One side of the great toe, for example, at the nail margin, can usually be anesthetized by 0 5 ml of 1 per cent procaine hydrochloride Toes are easily anesthetized by subcutaneous infiltration The amount of drug required is reduced by applying a rubber band at the base of the digit unless a peripheral circulatory deficiency is present, in which case application of a rubber band is contraindicated as inciting to gangrene (Miller and Markin, 1951, Miller and Harris, 1955, Miller, 1957)

Technique for Ankle Block—When deformity is extensive, especially when bone is involved, and when general anesthesia is contraindicated, ankle block anesthesia offers the best alternative To block the *deep peroneal nerve,* introduce the needle lateral to the tendon of the tibialis anticus and advance it until contact is made with bone Inject procaine hydrochloride, 10 ml of 1 per cent solution The *posterior tibial nerve* may be reached just medial to the tendo achillis Insert the needle through the deep fascia until it impinges on bone behind the medial malleolus Withdraw the needle slightly and inject from 5 to 10 ml of 1 per cent procaine hydrochloride Block the *sural nerve* by injecting from 5 to 10 ml of 1 per cent procaine hydrochloride on the lateral side of the tendo achillis Insert the needle horizontally until it strikes the bone and then withdraw it slightly Adjacent to each of the nerves is a large vessel which must be avoided so as not to introduce the drug directly into the blood stream

Surface Incisions of the Foot

The anatomic structures and skin cleavages of the foot run longitudinally (Figs 51 and 52) Even if the incision must be placed at a distance from the field of operation, it is usually better to have the incision follow alongside those structures A transverse incision is likely to cross a prominence of bone, so that the scar is subjected to the friction and pressure which are always especially experienced over such prominences Continuous friction and pressure over scar tissue may lead to an intractable problem Furthermore, the blood supply of the distal margin of a transverse incision is often impaired by the incision itself, so that healing is delayed It is true, however, that the scar of a longitudinal incision may restrict motion or produce contraction of the part

affects only the surface, disrupts only the skin margins, and produces only mild symptoms It is well to apply a dye type fungicide when trichophytic infection is anticipated (See Chapter 4, Infections of the Foot)

Weight Bearing.—Operations on the forefoot essentially involve connective tissues It takes from seven to ten weeks for connective tissue to heal sufficiently to withstand stress, therefore, tissues must be partly immobilized and free from maximum stress during the healing period Excessive weight bearing and the wearing of a shoe that has not been cut away at the area of the surgical wound to avoid restriction before healing has advanced may produce worse postoperative deformity than the original reason for operation About the third or fourth day, a cut-out Oxford may be worn over a heavy white sock This supports the foot without restricting the area of operation An uncut shoe should not be worn until edema and inflammatory process have subsided This takes from three to eight weeks A moderate amount of weight bearing may be permitted between twenty-four and seventy-two hours In three or four days the complete original dressing should be changed. At that time the sutures should be carefully examined Should there be signs of strangulation, the sutures should be removed, otherwise the sutures should be removed in about seven to ten days postoperatively If there is any gapping of the incision, a butterfly adhesive splint may be applied

The foregoing pertains primarily to surgical procedures of the forefoot Operations for tarsal and ankle deformities present special problems The parts must be immobilized for long periods and the aftercare is in accordance with individual needs

Complications of Foot Surgery

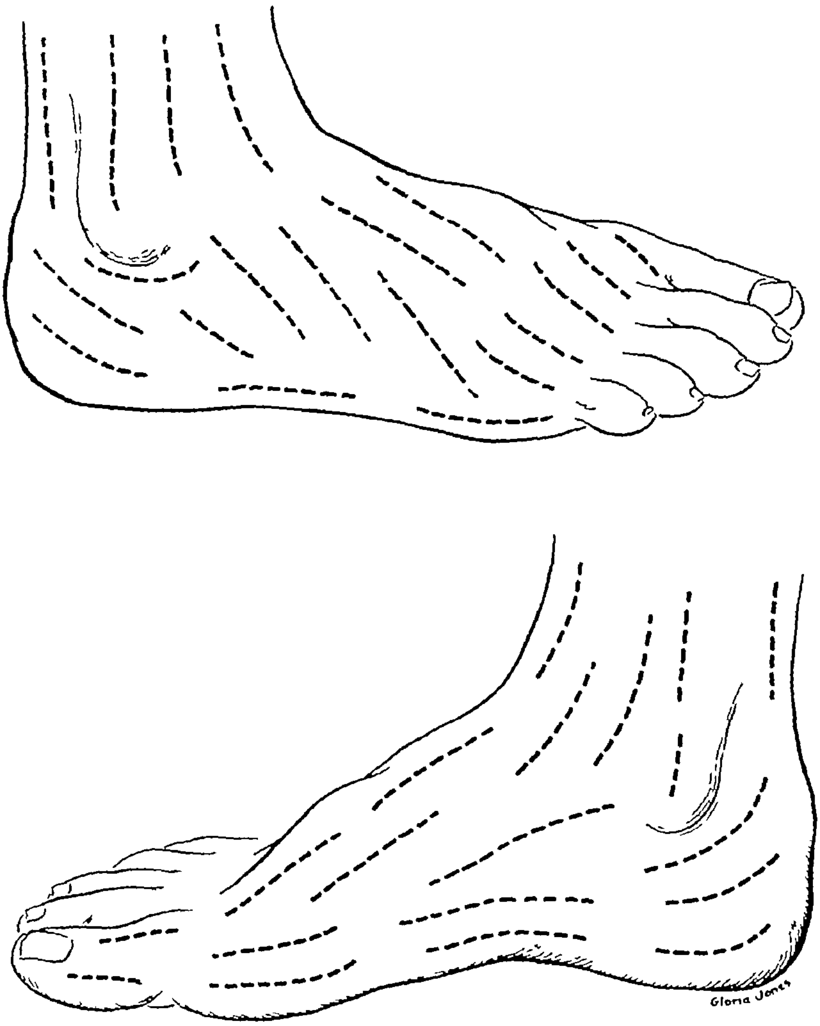
The possible complications to surgical procedures on the foot are (1) unequal weight distribution, (2) delayed healing, (3) recurrence of deformity or production of a new deformity, (4) allergic reaction to suture materials, (5) disruption of incision, (6) osteoporosis, (7) traumatic aneurysm, (8) postoperative anesthesia and paresthesia

Weight Distribution—Extensive surgery of the foot may permanently alter weight distribution When healing has been completed, that phase should be studied Weight bearing should be balanced as required by proper support, such as by inlays, wedging of shoes, or by application of bars on the sole of the shoes

Delayed Healing—Delayed healing is ordinarily due to any one or combination of the following factors vascular deficiency, excessive handling of tissues during surgical procedures, secondary infection, often tinea pedis, infections by *Staphylococcus aureus*, debilitating general diseases, and too early use or movement of the part The symptoms of infection appear within seventy-two hours The type of infection should be ascertained, so that suitable treatment may be instituted as early as possible

Infections by *Staphylococcus aureus* of surgical wounds appear to be increasing because of strains resistant to antibiotics Howe (1956) has called attention

A



B

Fig 52 – Skin cleavage *A*, Fibular side *B*, Tibial side

Postoperative Care

The patient should receive liberal sedation during the first twenty-four hours postoperatively The prophylactic value of antibiotics postoperatively has become controversial, because of the possibility of sensitization and also because of the development of penicillin-resistant strains of *Staphylococcus aureus* If the use of antibiotics is clearly indicated, early administration is mandatory If the presence of infection has been established, a broad-spectrum antibiotic should be ordered immediately As soon as the bacterial sensitivity has been ascertained, the particular antibiotic to which the organism is sensitive should supplant the broad-spectrum antibiotic

Tinea Pedis Infection – Spores of *tinea pedis* (*dermatophytosis,* also variously called *trychophytosis, Hong Kong foot, athlete's foot, epidermophytosis*) may invade the incision and interfere with healing This type of infection differs from a pyogenic infection in that the symptoms of a pyogenic infection are violent and the entire wound may be disrupted and exude pus Trichophytic infection

to carriers among hospital personnel, a situation underscoring the necessity for renewed emphasis on surgical asepsis Tachdjian and Compere (1957) are also among those of us who are worried about the reliance on antibiotics instead of strict antiseptic procedures

Recurrence or New Deformity.—An improper dressing and poor postoperative care may mean recurrence of the original deformity or production of a new deformity In such an eventuality, if correction is accomplished within the tenth postoperative day, the correction can be maintained. After that, the deformity may be permanent.

Allergic Reaction to Suture Material.—Occasionally an allergy to a suturing material that must be used subdermally produces a suture abscess The symptoms may not appear for weeks or months postoperatively. When they appear, the surface over the suture becomes soft and fluctuant and has a bluish discoloration On puncturing the area, a sanguineous material discharges. A circumscribed area of granulation has an aperture in the center through which the suture can usually be grasped for removal The area heals after removal of the suture or sutures, although occasionally the area must be reopened to remove the sutures

Disruption of the Incision.—Disruption of the incision may take place immediately following removal of skin sutures or a few days later. Disruption is due to poor healing quality which is commoner in the foot than in other parts of the body It may be due to hematoma in or underneath the incision, or it may be due to excessive handling of tissues during the operation

If disruption occurs, cleanse the wound with a mild antiseptic Coaptation of the skin margins by adhesive butterflies will hold the margins until union takes place.

Postoperative Osteoporosis.—An infrequent complication of foot surgery is a generalized osteoporosis of the bones in the area of operation Usually the entire foot becomes involved, although only one bone may be affected. The condition closely resembles Sudeck's atrophy (See page 305) A definite cause is not known The symptoms appear about six to eight weeks after an apparently uneventful postoperative course. The disease manifests itself by pain, swelling, and redness of the foot, beginning without apparent cause until roentgenograms of both feet disclose loss of bone substance in one or more bones (Fig 53)

Traumatic Aneurysm.—Aneurysm as a complication of surgery of the foot is uncommon. When it happens, it is due to injury to a vessel during surgical procedures, especially when a tourniquet has been applied If an artery has been completely divided, it will retract in its sheath and close with little ill effect, however, if it has been only nicked and not divided, a hematoma forms and eventually becomes a false aneursym (Fig 54). If an adjacent vein is also nicked, development of an arteriovenous aneurysm may be anticipated. Scott (1955), Webb-Jones (1955), and Coughlin (1951) have reported such cases. In most cases of aneurysm a second surgical procedure is necessary to extirpate the aneurysm and ligate the vessels

Postoperative Anesthesia; Paresthesia; Causalgia.—Anesthesia or paresthesia is a complication sometimes encountered after a surgical procedure performed on the foot It happens when a cutaneous nerve in the path of operation has been

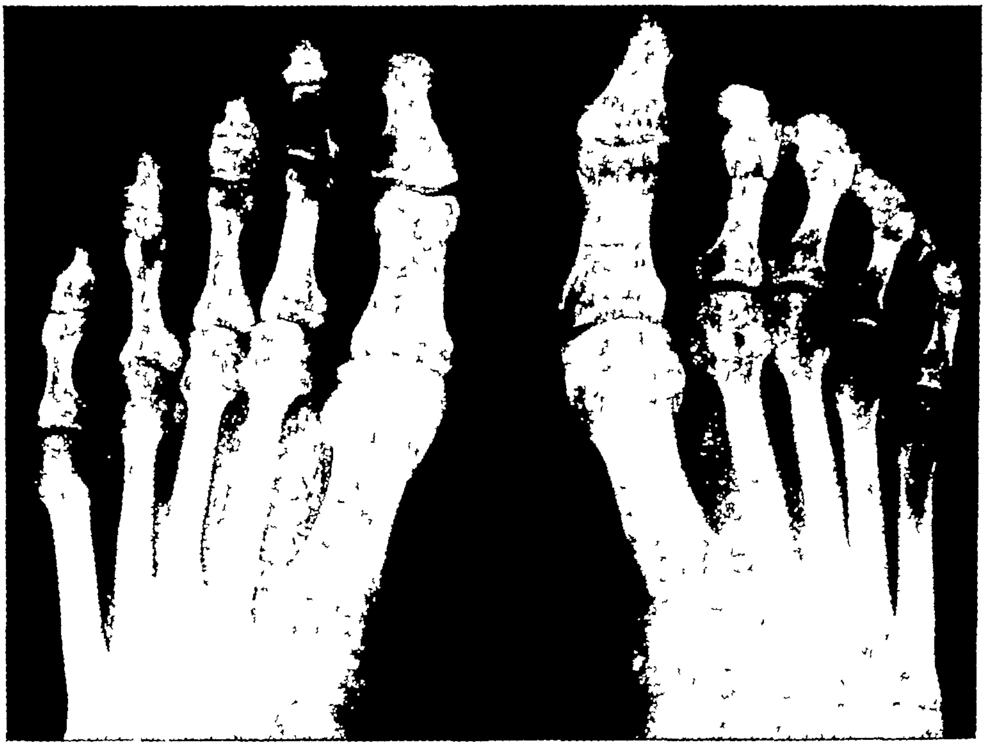


Fig 53 —Postoperative osteoporosis of right foot Left foot normal

Fig 54 —Traumatic aneurysm of posterior tibial artery following operation along medial malleolus (Courtesy Dr Ronald Tanner)

CHAPTER FOUR

Infections of the Foot

PYOGENIC INFECTIONS

SUPERFICIAL PYOGENIC INFECTIONS ARE OFTEN INDUCED OR AGGRAVATED BY FOOTgear The terminus of the toes makes the toes susceptible to a *felon,* a type of osteomyelitis, occurring also in the fingers The troublesome ubiquitous wide variety of tinea pedis may combine with some other invading organism Severe infections of the foot result from neglected minor scratches, irritations from friction, or contamination as by scissors Treatment for gross injury is sought promptly, whereas treatment for minor irritations and inflammations is likely to be delayed The patient continues to wear shoes and bear weight until the infection extends into the fascial planes, tendon sheaths, or lymphatic channels.

Pyogenic organisms may invade any part of the foot through an abrasion in the surface The abrasion may be microscopic and the patient completely unaware of its presence

Cellulitis

When pyogenic bacteria, usually staphylococci, enter the numerous irritated hair follicles, diffuse cellulitis results Continued irritation and friction by the eyelets of the shoe over the dorsum of the first metatarsal cuneiform joint, which is generally the highest point on the dorsum of the midfoot, in the presence of pyogenic organisms may produce extensive violent cellulitis of the dorsum of the foot

Cellulitis is treated by complete rest, continuous hot fomentations, and antibiotics taken internally If organization takes place, free drainage must be induced

severed or injured The patient reports anesthesia a few days postoperatively In most instances, sensation returns in three or four months, in a few instances, patients become accustomed to loss of sensation and are not troubled by it In rare cases, the proximal end of a severed nerve, or the whole nerve, may become incorporated in scar tissue, which produces causalgia (See Chapter 13)

REFERENCES

Bunnell, Sterling Surgery of the Hand, ed 2, Philadelphia, 1951, J B Lippincott Co , p 5

Coughlin, J J Arterio-Venous Aneurysm of Foot Following Plantar Fasciotomy, Northwest Med **50** 772-773, Oct , 1951

Dettinger, G B , and Bowers, W F Tissue Response to Orlon and Dacron Sutures, A Comparison With Nylon, Cotton and Silk, Surgery **42** 325, Aug , 1957

Howe, C W Prevention and Control of Postoperative Wound Infections Owing to Staphylococcus Aureus, New England J Med **255** 787, Oct , 1956

Miller, D S Gangrene From Arterial Injuries Associated With Fractures and Dislocations of the Leg in Young and in Adults With Normal Circulation, Am J Surg **93** 367-375, March, 1957

Miller, D S , and Markin, Leo Vascular Complications in Fractures and Traumatology, Indust Med & Surg **20** 323-324, July, 1951

Miller, D S , and Harris, A J Gangrene in the Aged Associated With Fractures of the Extremities, J Internat Coll Surg **24** 669-688, Dec , 1955

Partipilo, A V Surgical Technique and Principles of Operative Surgery, ed 6, Philadelphia, 1957, Lea & Febiger, pp 125-142

Pick, J F Surgery of Repair, Philadelphia, 1949, J B Lippincott Co , pp 1, 109

Scott, J H S Traumatic Aneurysm of the Peroneal Artery, J Bone & Joint Surg **37B** 438, Aug , 1955

Tachdjian, M O , and Compere, E L Postoperative Wound Infections in Orthopedic Surgery, Evaluation of Prophylactic Antibiotics, J Internat Coll Surg **28** 797-805, Dec , 1957

Tauber, Robert Basic Surgical Skills, Philadelphia, 1955, W B Saunders Co

Webb-Jones, A Aneurysm After Foot Stabilization, J Bone & Joint Surg **37B** 440, Aug , 1955

bolic infection of bone gives rise to hematogenous osteomyelitis in which the causative organism is more specific The disease is prevalent in children, especially boys, from 5 to 14 years of age. The bones of the foot may be involved, the tibia is often affected. In growing children, the growth of the bone may be arrested, so that discrepancy in length of the legs results. Pain over the infected area, swelling and induration of the soft tissues, and signs of general toxicity comprise the symptoms.

Acute Osteomyelitis.—In *acute hematogenous osteomyelitis,* systemic reactions are severe Roentgenograms reveal medullary cloudiness and periosteal thickening, osseous changes may or may not be present, depending on the severity and duration of the disease

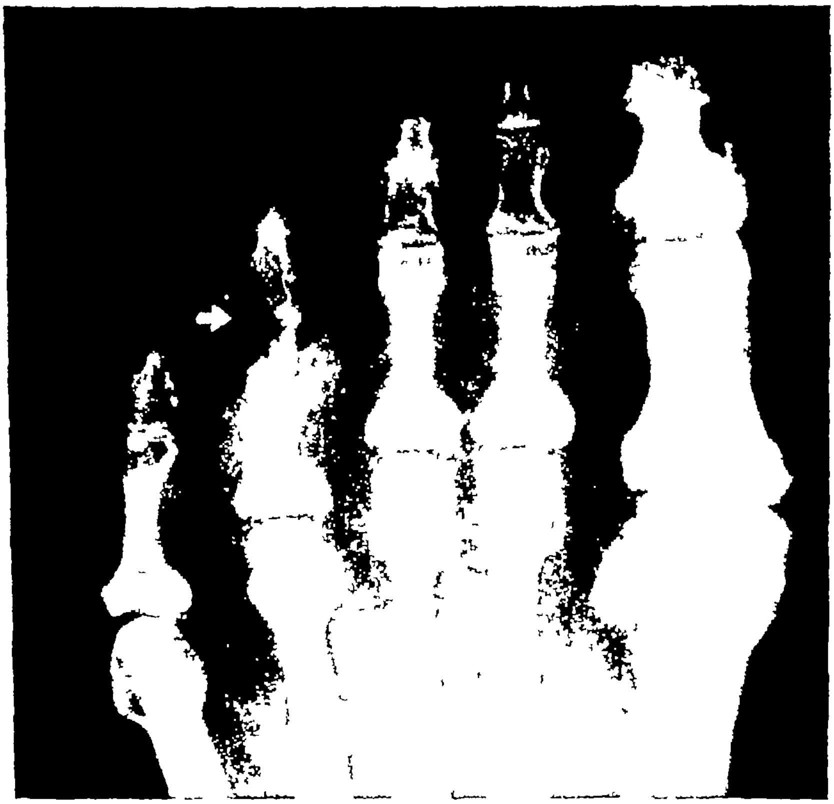


Fig 55—Arrow points to site of acute osteomyelitis of head of fourth proximal phalanx

Wilson and McKeever (1936) reviewed the cases of sixty-four patients with ninety foci of infection, of which the distribution regarding the foot was as follows calcaneus, five, metatarsals, three, phalanges (Fig 55), two

Chronic Osteomyelitis.—*Chronic osteomyelitis* frequently follows the acute form The symptoms are milder but prolonged. When healing begins after the acute stage, portions of dead bone which harbor bacteria may persist and gradually detach themselves as sequestra, or, in a comminuted fracture, a small fragment of bone may resist union during the healing process and sequestrate The borderline of the sequestration is encased by periosteal new bone representing the healing reaction and is known as the *involucrum* The soft tissues are moderately swollen, greatly indurated, and adherent. In prolonged cases, scar tissue

Lymphangitis

Lymphangitis is an acute infection of the lymphatic channels caused by entrance of staphylococci or streptococci through a small wound or abrasion The abrasion may be trivial, but the infection, once it enters the lymphatic channels, spreads rapidly Throughout the entire surface integument there is a fine mesh network of lymph channels which lead into main trunks The trunks are filtered by lymph glands along their course In most infections through abrasions, sepsis is contained locally and its course is mild and benign It is only when a virulent pyogenic organism succeeds in entering the lymphatic system that lymphangitis ensues

Symptoms.—The onset is sudden There may or may not be a history of injury or abrasion Pain and tenderness are experienced over the focal point, a rapidly rising temperature often follows chills In twelve to twenty-four hours bright red streaks appear along the lymphatic channels, inflammation and edema surround the streaks The lymph glands proximal to the focus of infection become swollen, painful, and inflamed Symptoms of general toxicity may be severe, out of all proportion to the appearance of the local lesion

In a few cases, organization of pus takes place at the focus of infection Occasionally the infection spreads rapidly despite heroic measures until general septicemia develops Death then ensues in a few days Koch (1934) has contributed a fine study of the subject as it applies to the hand

Program of Treatment Surgical Procedure.—Under treatment by early administration of antibiotics and chemotherapy, infections subside Patients having lymphangitis should be hospitalized and kept at complete rest The leg should be elevated Massive fomentation packs, covering the entire foot and leg, should be applied continuously High blood levels of antibiotics or sulfonamides or both these agents should be maintained Whenever possible the organism's sensitivity should be ascertained and the appropriate antibiotic instituted Close attention should be paid to complications of toxemia, such as dehydration and imbalance of potassium and sodium

Incision for drainage is rarely indicated, it may even be harmful (Kanavel, 1925, Koch, 1929) Even when there is evidence of organization, it is better to err on the conservative side by not incising The only time that an incision is indicated is when there is evidence of pus under the surface That evidence is made known by the presence of a blister or fluctuation directly under the skin, ordinarily at the focal point of infection but, occasionally, anywhere along the course of the lymphatic channels

Osteomyelitis

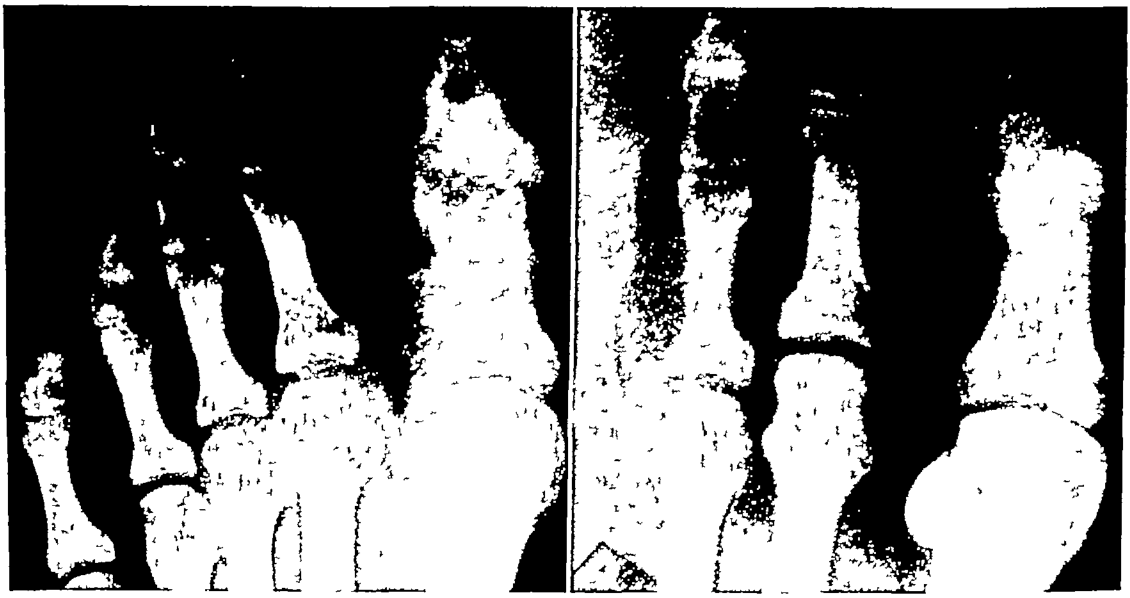
Osteomyelitis is an acute or chronic, local or diffuse infection of the marrow The term *pyogenic osteomyelitis* designates an infection of bone caused by a pyogenic organism, usually of simple type, such as staphylococcus or streptococcus The infection may be due to direct extension or it may be embolic Infection of bone by direct extension from surrounding tissue takes place in compound fractures, from ulceration of soft tissues, and in cases of septic amputations Em-

Fig 57 —Felon of great toe

Fig 58 —Osteomyelitis of first distal and head of proximal phalanges after injury

forms, contracts, and causes puckering of the soft tissues A sinus leading from the skin surface to a sequestrum of necrotic bone discharges a purulent material Roentgenograms show osteolytic and osteosclerotic changes as well as distortion of the outline of the bone (Fig 56)

Since the advent of the antibiotics, the acute stage of the disease is not so difficult to treat, and surgical intervention is less frequently indicated in cases of direct extension of acute infection The principles governing treatment of acute hematogenous osteomyelitis are essentially the same as for treatment of pyogenic infection of other tissues (1) early diagnosis, (2) prompt antibacterial treatment, and (3) correctly timed surgical drainage



A B

Fig 56 —*A*, Osteomyelitis of entire hallux *B*, After healing

Chronic osteomyelitis is an inevitable surgical problem The sensitivity of the organism recovered in drainage is determined preoperatively Adequate antibiotic therapy is begun at least four days before and continued for two weeks or longer after operation Roentgenographic studies are necessary to ascertain whether there is sufficient involucrum to form a support after all sequestra and necrotic bone have been excised Sequestra may vary from minute particles to the entire circumference of the shaft of the bone All necrotic bone must be excised, including any sinuses that connect the area of sequestration with the surface In most cases the wound may be closed primarily and the extremity immobilized in a plaster cast until completely healed A window over the wound permits change of dressings

Felon.—A *felon*, or *whitlow*, is a septic infection of the pulp space of the distal phalanx of a finger or toe (Figs 57 and 58) Pus collects in the pulp space and haversian canals of the epiphysis of the phalanx, distending the enclosed space The pressure thus produced deprives the bone of blood to the tip of the phalanx, thereby causing necrosis and sequestration of the bone

always secondary to a tuberculous focus elsewhere in the body, this focus should be disclosed. A pulmonary lesion adds signally to the gravity of the prognosis.

Tuberculosis of the bones of the foot may occur at any age. As a rule, the chronic infection is fairly widespread, although isolated foci may be found, particularly in the calcaneus and talus

According to Miltner and Fang (1936), in multiple infections the most important weight-bearing bones are affected, the order of incidence being, from the highest to the lowest, as follows calcaneus, talus, first metatarsal, navicular, and first and second cuneiform bones Cozen (1951) reported a case of tuberculosis of the heel of a 2-year-old child in which reactions to tuberculin tests were repeatedly negative, yet the disease proved present on biopsy.

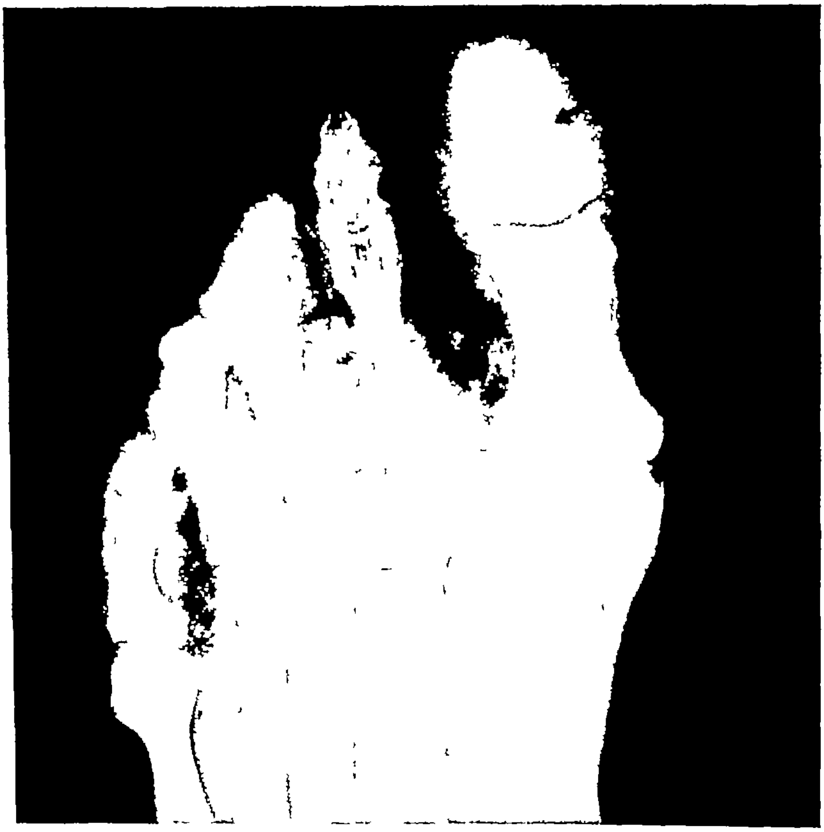


Fig 59 —Osteomyelitis of first distal phalanx after operation on nail

Complete excision or curettage of a localized tuberculous focus and adequate antibacterial therapy have been consistently successful If a bone or bones can be removed in entirety without producing excessive deformity and disability, excision may be a curative measure Such a procedure, however, is limited almost entirely to the bones of the foot and hand

In general, tuberculosis should be suggested in the presence of an unrelenting prolonged chronic destructive process in the bone, with recurring acute exacerbations involving all or part of a bone, provided the commoner bone and joint diseases have been ruled out Reeves (1958) presents a thorough discussion of differential diagnosis of tuberculosis of the talus in a case in which the clinical diagnostic impressions included arthritis, degenerative disease, and granuloma-

Symptoms—A day or two after the onset of infection the distal part of the toe becomes indurated, swollen, and throbbing Pain is violent, sleep is impossible The discrepancy between objective observations and the severity of pain leads to the diagnosis of a *felon* After several days, pain diminishes as necrosis of bone sets in, only the roentgenographic examination discloses the presence of *osteomyelitis of the distal phalanx*

Treatment—After the infection has been controlled, if the bone of the distal phalanx has sequestrated completely, it is sometimes advisable to leave the sequestrum in position, in the hope that an involucrum and new bone will form about it and that it will serve as a scaffold In most cases, however, it is far better to remove all sequestrated bone (Compere)

Procedure—Under general anesthesia the pulp space should be widely opened by a semicircular (fishmouth) incision, extending from one side of the toe to the other side and encircling the distal half of the distal phalanx Secondary vertical incisions are made into the pulp space to facilitate drainage If sequestra are present, either they should be removed or else the necrotic parts should be gently curetted and an antibiotic applied locally In most cases the process subsides in two or three weeks and the bone regenerates

Sometimes infection progresses so rapidly that amputation of the distal phalanx or of the entire digit becomes necessary In such instances the skin flaps should be left open for free drainage

INFECTIONS OF PARTICULAR SITES ON THE FOOT

Bailey (1957) has discussed the subject of infections of the foot largely in terms of infections in particular areas of the foot infected adventitious bursa associated with a corn, infected bursa over a hallux valgus, infection of terminal pulp space (Fig 59), suppurative tenosynovitis, infection of interdigital subcutaneous spaces, the heel space, the web spaces, the deep fascial spaces of the sole, the central, medial, and lateral plantar spaces, the dorsal subcutaneous and the dorsal subaponeurotic spaces He also includes brief consideration of infected blister and of paronychia in the lateral region of the nail, which is especially common in the great toe as a complication of so-called ingrown nail

OSSEOUS TUBERCULOSIS OF THE FOOT

The bones of the foot are seldom the sites of tuberculous lesions, but the possibility must not be overlooked in establishing the diagnosis Cozen (1951) reported a case of tuberculosis of the right calcaneous in which the lesion was proved by biopsy after negative reactions to the tuberculin skin test

Since the advent of antibiotics, especially streptomycin, tuberculosis of the bone is infrequent When it is encountered, it may be difficult to diagnose because it may simulate other diseases of bone A positive reaction to the tuberculin skin test may or may not rule out the disease

Tuberculosis of bone and joints is a slow, unrelenting, destructive manifestation of a systemic disease induced by the tubercle bacillus Since the lesion is

show a more or less circumscribed area of rarefaction of the bone. The cortex shows increased density, proliferative changes, and periosteal thickening (Fig 61)

The abscess should be opened and curetted and all necrotic and overhanging bone margins removed In some cases the wound may be closed primarily, but if the cavity contains a great deal of purulent material, it is safer to pack it with petroleum-saturated gauze for free drainage When all signs of infection have subsided, a secondary closure may be attempted

SCLEROSING NONSUPPURATIVE OSTEITIS OF GARRÉ

Garré, in 1893, first described sclerosing nonsuppurative osteitis, which is a chronic nonspecific inflammation of bone, most frequently involving the tibia, but it may involve any long bone, especially of the foot, and usually affects an entire single long bone The cause is not certain The disease is believed to be due to a low-grade embolic infection transmitted to the bone by the blood or lymphatics

Symptoms.—The onset is insidious and characterized by pain and swelling over the entire bone, toxic symptoms depend on the bone involved If, as is often the case, the tibia is infected, general symptoms may be severe, however, when such a small bone as a first proximal phalanx or metatarsal (Fig 62) is affected, symptoms may not be generalized The disease begins subacutely and gradually becomes chronic Occasionally, however, the disease begins with acute and alarming symptoms

Diagnosis—Progressive pain and swelling over a long bone without traceable history or cause are characteristic Roentgenograms reveal increased density of the bone and thickening of the cortex because of the laying down of new bone under the periosteum and in the wall of the medullary canal, which are the important clues to diagnosis Because similar observations are consistent with osteoid osteomas, roentgenograms in multiple planes are necessary to ascertain whether a nidus is present The presence of a nidus justifies a tentative diagnosis of osteoid osteoma

Treatment—Radiation therapy and therapeutic dosages of antibiotics given over a period of weeks are effective Surgical treatment consists of excision of a portion of the cortex of the bone to expose the medullary canal If necrotic areas are encountered, they are curetted, an antibiotic may be implanted and the wound closed with or without drainage

COCCIDIOIDAL INFECTIONS

Grebe (1954) reports a case of monostatic coccidioidal infection in which the bone lesion was in the left calcaneus Evidence of disease or injury had not been observed There was no response to wide excision of the bone lesion, but rapid healing was accomplished by treatment with 2-hydroxystilbamidine When coccidioidal infections disseminate, the outcome is usually fatal

McMaster and Gilfillan (1939) reported twenty-four cases of *coccidioidal osteomyelitis* with multiple foci, involving various parts of the body, including

tous proliferation Reeves emphasizes a sign which is almost pathognomonic roentgenographic evidence of partial sclerotic reaction, indicating unsuccessful healing, and the fragmented talus more severely diseased in one part than otherwise Reeves calls attention likewise to the need for follow-up roentgenograms, because the first ones are likely to appear normal

BRODIE'S ABSCESS

Brodie's abscess is a localized infection in the shaft of a long bone, most commonly involving the lower end of the tibia (Fig 60) About half of the reported cases have been in this area It is generally agreed that the abscess is caused by staphylococci of low virulence It happens mostly during adolescence, boys being affected more in a ratio of five to one The abscess may be a residual or complicating feature of acute osteomyelitis The abscess may be sterile In many cases onset is acute, the symptoms rapidly subsiding, but for months or even years mild pain and fusiform swelling of the area may persist Roentgenograms

Fig 60 Fig 61

Fig 60 —Arrow points to Brodie's abscess of lower end of tibia

Fig 61 —Brodie's abscess of third metatarsal Note increased density and periosteal thickening of entire metatarsal shaft

and shows clinical and roentgenologic changes similar to those in chronic osteomyelitis Treatment is essentially the same as for chronic osteomyelitis.

DERMATOPHYTOSIS

Dermatophytosis or *tinea* infection, commonly called *ringworm*, is almost always present on the foot Spores of one or more strains of trichophytes are commonly harbored between the toes and under the nails but may invade any part of the foot The spores may lie dormant for months or years before they mature and multiply under conditions, favorable to them, of excessive moisture, friction, pressure, or trauma, either accidental or iatrogenic When the disease

Fig 63

Fig 64

Fig 63 —Acute dermatophytosis of toes
Fig 64 —Dermatophytosis in plantar creases of toes

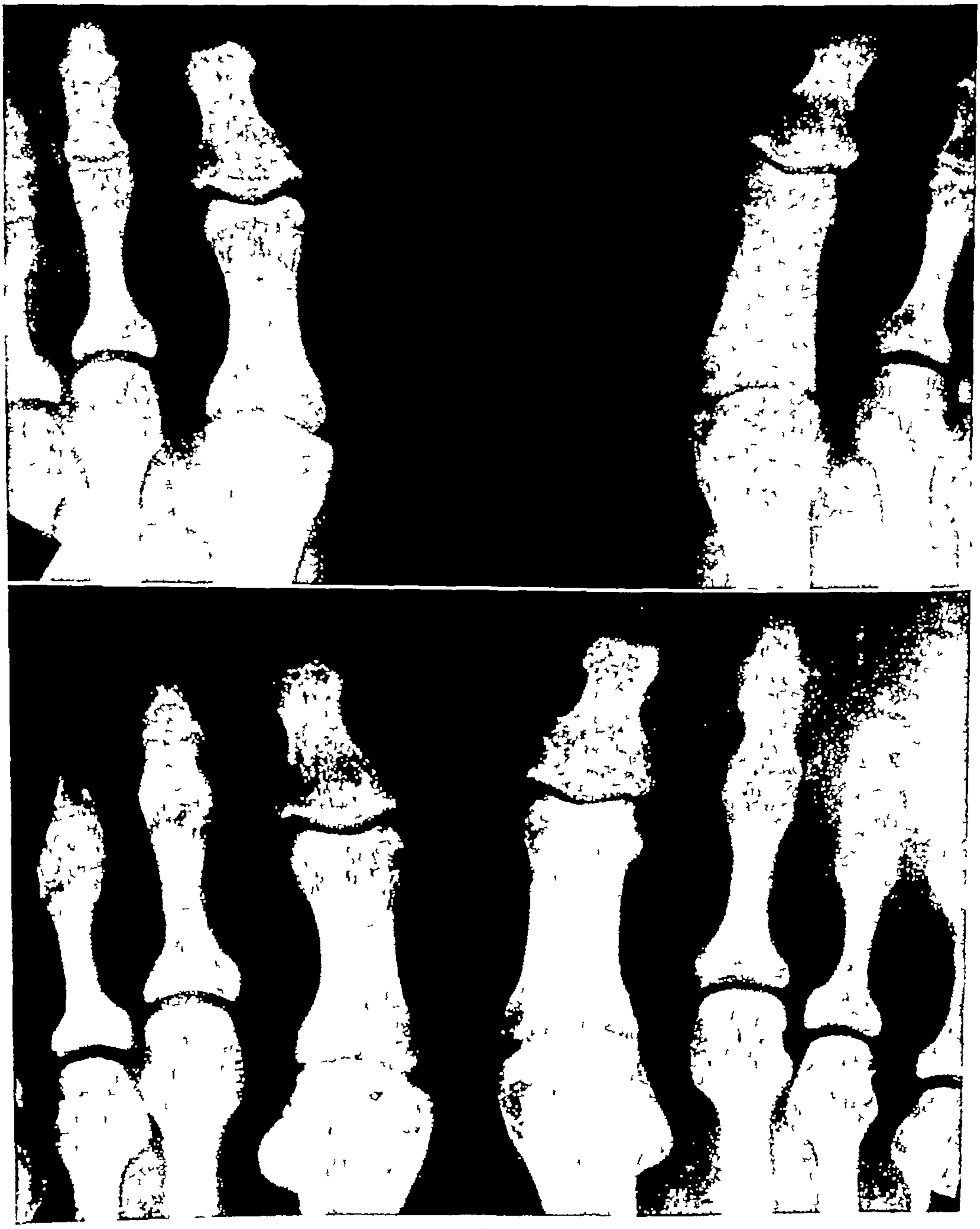
is active, it assumes various forms and degrees, from minute fissures between the toes to a vesicular scaly dermatitis over large areas on the entire foot (Figs 63 and 64), and from a low-grade inflammatory process to a violent infection, although serious illness is rare Dermatophytosis is scarcely ever cured

One theory is that in many cases of thrombophlebitis, the causative pyogenic organism may have found entrance in a fissure caused by trichophytosis between the toes Such a fissure may be microscopic Instances of retardation of healing of a wound in the foot may be due to the invasion of dormant fungi into the wound According to some observers, fungi may even act to make certain organisms penicillin-resistant

the foot The average age in their cases was 32 years, predominantly men, thirteen of the patients died, all of whom were proved to have had pulmonary involvement

The disease is caused by a specific fungus, *Coccidioides immitis,* which gains entrance through the respiratory tract or skin and is disseminated through the blood or lymph channels or by direct extension The disease is usually chronic

A.



B

Fig 62—*A,* Osteitis of Garré Left, Normal foot for comparison Right, Sclerosing and periosteal thickening of first proximal phalanx *B,* One year later Note decrease in bone density and disappearance of periosteal thickening

fungus resembling Actinomyces The disease begins as a granuloma, generally on the sole, then massive swelling of the foot takes place. New tumors form while old ones soften, and the foot increases in size enormously, becoming deformed Franz and Albertini (1954) found extensive osteoporosis of the tarsal bones in one of their patients

No specific treatment is known. Dixon (1941) believes that antibiotics may prove effective in advanced cases In intractable cases, amputation above the diseased area may be indicated.

REFERENCES

Baer, R L, Rosenthal, S A, Litt, J Z., and Rogachefsky, H Experimental Investigations on Mechanism Producing Acute Dermatophytosis of Feet, J A M A 160 184-190, Jan 21, 1956

Bailey, Hamilton Infections of the Foot, J Internat. Coll Surg 27 475-482, 1957

Compere, E L Personal communication to author

Cozen, Lewis Osseous Tuberculosis With Negative Tuberculin Test, J Bone & Joint Surg. 33A 797-800, July, 1951

Dixon, J M Sulfanilamide Therapy in Madura Foot, Virginia M Monthly 68 281-282, May, 1941

Dolce, F A Shoe Dermatitis, Among Soldiers, Mil Surgeon 95 505, 1944

Fang, H C, and Miltner, L J Periostitis of the Os Calcis, J Bone & Joint Surg 16.355-364, 1934

Franz, A, and Albertini, B Primitive Mycetoma of the Foot, Madura Foot (Sul micetoma primitivo del piede piede di Madura, Chir org movim 40.412, 1954) abstracted in Internat Abstr Surg, p 196, Feb, 1956

Garré, C Ueber besondere Formen und Folgezustande der akuten infestiosen Osteomyelitis, Bruns Beitr klin Chir 10 241-298, 1893

Gaul, L E, and Underwood, G B Primary Irritants and Sensitizers Used in Fabrication of Footwear, A M A Arch Dermat & Syph 60.649-675, Nov, 1949

Grebe, A A Monostotic Coccidioidal Infection, Report of a Case Successfully Treated With 2-Hydroxystilbamidine, J Bone & Joint Surg 36A·859-862, July, 1954

Kanavel, A B Infections of the Hand, Philadelphia, 1925, Lea & Febiger

Koch, S L Felons, Acute Lymphangitis and Tendon Sheath Infections, Differential Diagnosis and Treatment, J A M A 92 1171-1172, April 6, 1929

Koch, S L Acute Rapidly Spreading Infections Following Trivial Injuries of the Hand, Surg Gynec & Obst 59 277-308, Sept, 1934

McMaster, P E, and Gilfillan, Charles Coccidioidal Osteomyeltitis, J A M A 112 1235-1237, April 1, 1939

Miltner, L J, and Fang, H C Prognosis and Treatment of Tuberculosis of the Bones of the Foot, J Bone & Joint Surg 18 287-296, April, 1936

Niles, H D Dermatitis Due to Shoe Leather, J A M A 110 363-365, Jan 29, 1938

Reeves, J D Differential Diagnosis in Case 44122 Presentation of Case, New England J Med 258 612-615, March 20, 1958

Wilson, J C, and McKeever, F M Bone Growth Disturbance Following Hematogenous Acute Osteomyelitis, J A M A 107 1188-1192, Oct 10, 1936

Special precautions in cleanliness and generous use of fungicides preoperatively and postoperatively reduce the incidence of morbidity from surgical procedures on the foot

Dermatophytosis is generally thought to be contagious, however, Baer and his associates (1956), in an experimental attempt to infect sixty-eight subjects proved to be free of the disease, were not able to infect a single patient

DERMATITIS FROM SHOE IRRITANTS

Niles (1938) reported two cases of severe dermatitis of both feet which was first assumed to be due to tinea infection but later was proved conclusively to have been due to allergens in the shoe leather

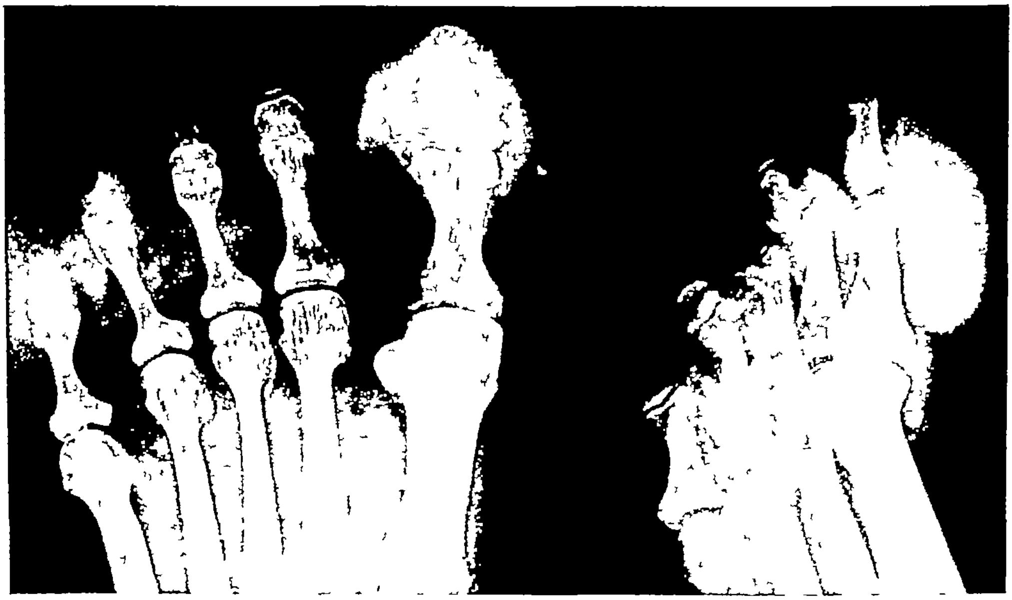


Fig 65—Massive granuloma of great toe Result of mycotic infection acquired in tropical country

Gaul and Underwood (1949) made an exhaustive study of 160 cases of dermatitis pedis which they traced to allergens in various fabric and leather parts of shoes The onset of dermatitis varied from a few hours to seven months after the offending shoes had been worn The allergens affected persons of all ages, including a child, aged 16 months

Many of the chemicals employed in processing leather or fabrics of which shoes are made may cause dermatitis, not necessarily on an allergic basis but because they are irritants Dolce (1944), in a study of cases of shoe leather dermatitis, found chromic acid and potassium dichromate to be the commonest irritants

MADURA FOOT (MYCETOMA)

Mycetoma is a chronic granulomatous disease (Fig 65), mainly found in tropical countries, especially in certain districts of India It is caused by a

Treatment.—The treatment for chemical and x-ray burns is as follows.

Chemical Burns.—For local and immediate treatment of an acid burn, pour over the area a neutralizing agent, such as a solution of sodium bicarbonate, for an alkali burn, diluted vinegar. Do not attempt to take off clothing but rather cut away all clothing. Remove those foreign bodies that can be removed without disturbing the wound. The burn wound is not infected when first seen. Infection is likely to be introduced by excessive and contaminating handling, therefore, gentle cleansing and application of a sterile emollient compression dressing are advised.

Fig. 66

Fig. 67

Fig. 66.—Severe plantar contraction of toes resulting from third degree burn.
Fig. 67.—Destruction of calcaneal tuberosity by third degree burn.

CHAPTER FIVE

Burns and Freezing; Foreign Bodies and Gunshot Wounds; Sprains and Athletic Injuries

BURNS AND FREEZING

A COMPREHENSIVE DISCUSSION OF BURNS AND FREEZING IS OUTSIDE THE SCOPE OF this text, however, because of the high incidence of frostbite of the foot and because burns of the foot from irradiation are not uncommon, at least brief recognition of the injuries is in order

BURNS

Burns are caused by exposure to intense heat, by contact with strong chemicals or live electricity, or by overexposure to roentgen rays or radium It is only the burn from irradiation that is likely to come to the attention of the foot surgeon Sudden burns of the foot are rare because of the protection given by shoes (Figs 66 and 67) When they do occur during industrial accidents, they are in most instances cared for by industrial surgeons

Classification—According to the depth of the burn, burns are primarily classified as first degree, meaning redness, second degree, meaning blistering, and third degree, meaning charring to the bone Unfortunately, it is difficult to determine the depth of a burn on first examination A broad division may be made between those burns that heal without scarring, and those that leave extensive scars To be sure, classification and meaning have been reduced to oversimplified terms.

Fortunately, most burns are not deep, but they are nevertheless painful because burning destroys the protective covering of the nerve endings in the epidermis

superficial freezing, however, Vinson and Schatzki (1954) observed roentgenologic changes in the bone resulting from frostbite Edwards and Leeper (1952) analyzed seventy-one cases of frostbite of the extremities, in which all showed necrosis, the extent of which was in direct relation to duration of freezing after onset of symptoms Chronic vasospasm or hyperhidrosis, history of cold injury, wounding, and possibly smoking were thought to be personal contributing factors Later or residual results of severe freezing can produce formidable problems of necrosis and its consequences (Lewis, 1941) Blair and his associates (1957) studied 100 cases of freezing, in which the persistent symptoms were cold feet, numbness, pain, hyperhidrosis, deformed nails, scarring, and mutilation of terminal phalanges

The basic principle is that initial therapy should be conservative The application of extreme heat may be harmful, because it causes vasoparalysis and consequent stasis and transudation of plasma, but relative warmth is advisable. The old theory that a frozen limb must be thawed out slowly has been repeatedly disproved, although Edwards and Leeper (1952) have suggested that more data are needed for conclusion The consensus seems to be that rapid thawing is advisable, provided the agent is no warmer than about 40° C (104° F)

Strict cleanliness and asepsis must be observed for six to ten days to prevent secondary infection Treatment must be expectant during this period which will determine whether destruction will terminate in gangrene of the part and whether a line of demarcation will form During the waiting interim, judgment must be informed regarding the advisability of vasodilation by means of drugs, such as heparin, or by sympathectomy, according to which course may prevent gangrene When gangrene threatens, the part must be amputated

Chilblains (Perniones).—Repeated mild frostbite produces vasomotor instability resulting in chilblains, or perniones The condition is characterized by recurrent attacks of hyperemia, burning, and tenderness When the foot is affected, the symptoms are mostly over a bony prominence, especially over the lateral side of the head of the fifth metatarsal

Treatment is essentially mechanical and negative the avoidance of pressure over the part by padding or shoe appliances and the avoidance of further chilling

Immersion Foot.—It was not until World War II that immersion foot was given serious attention (Fausel and Hemphill, 1945) Webster and his colleagues (1942) studied 142 cases of long submersion of the feet in cold water The limbs had been immobile and constricted by boots The result was comparable to so-called trench foot and shelter foot and to ordinary frostbite

The traumatizing symptoms on removal from the water were increased and rapid swelling as the feet became red and hyperemic and the temperature of the part became extremely elevated, although without sweating, strong pulses in the vessels of the feet, livid cyanosis, blebs and extravasation of blood, ecchymosis, vasodilation and vascular wall impairment, especially over the medial aspects of the first metatarsophalangeal joint and the longitudinal tarsal arch There were many degrees and patterns of anesthesia, hypesthesia, and paresthesia

Kaye (1956), in a comprehensive outline for treatment of burns which is recommended for study, has this to say, in part, regarding local treatment:

> Local and systemic treatment should be carried out simultaneously.
>
> Local drugs have little effect upon the healing of a burn and often prove detrimental. When drying of the wound has been achieved and infection controlled, the treatment of a full-thickness burn becomes a surgical problem of skin replacement.
>
> Local treatment of burns has gone through many phases: wax, tannic acid, pressure dressings, and the exposure method. . . . [The occlusive pressure dressing (Allen and Koch, 1941) has been successful. Its] objective is to cover the open wound with the simplest possible dressing that will protect it from reinfection, provide for drainage of serum, exert a uniform pressure, and be easily removed if infection develops.*

Kaye lists the following aims in the treatment of burns:

> (1) prevention and treatment of shock, (2) prevention and treatment of infection, (3) utilization of a local therapy that will insure drying of the burn wound and permit early skin grafting, (4) maintenance of adequate nutrition and hemoglobin level during the healing stage, and (5) prevention and correction of contractual deformities.*

X-ray Burns.—Roentgen-ray burns are manifested from six months to a year after excessive irradiation. They are common on the plantar surface of the foot because of extensive use of roentgen therapy for verrucae plantaris. (See Intractable Plantar Keratosis, page 184.) Roentgen-ray burns are resistant to all forms of therapy; however, the area should be kept clean and free from all pressures so as to stimulate healing and growth of skin. In many cases grafting of new skin is necessary. (See page 202.)

FREEZING

Response to cold is individual and variable and therefore unpredictable. Duration and severity of exposure alter the clinical appearance. In general, at first the area becomes blanched or white in response to the cold. As exposure continues, the area may become stiff and brittle. Freezing produces local sequelae similar to those of burns, which may be grouped as *cryopathies* and include frostbite, actual freezing, immersion foot, and the almost identical conditions referred to as *trench foot* and *shelter foot*. Freezing may be divided into three degrees of injury. The first degree involves only the epidermis; the second and third degrees involve the subcutaneous tissues. Frost injuries produce pathologic changes of essentially two types: those due to vasomotor disturbances caused by exposure to cold and those due to pathologic changes caused by actual freezing of the blood vessel walls or of their blood contents. If the skin has been frozen, the following effects are invariably detected: (1) local and active dilatation of the minute vessels, (2) surrounding flush caused by an arteriolar dilatation, (3) local whealing of the skin and blistering when freezing has been extreme.

Frostbite.—Frostbite represents a borderline condition between actual freezing and immersion foot (Bigelow, 1942; Brownrigg, 1943). Frostbite implies

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*From Kaye, B. B.: Burns: An Outline for Treatment, Am. J. Surg. 92:123-138, July, 1956.

Fig 69 —Part of needle in great toe Presence of foreign body dormant for a long time, suddenly became symptomatic

Fig 70 —Part of needle in foot Acute symptoms immediately after entrance

Treatment was by dry cooling and refrigeration by application of icebags, exposure to a fan, and then slow dry cooling at room temperature with elevation of the feet Patients were comfortable within a few hours, blebs resorbed without breaking, and the average hospitalization was about a month, although minor symptoms may have persisted for a couple of years

Ungley (1943) suggests that massage is contraindicated and that active movements aid circulation, although walking or positions restricting circulation are dangerous, body warmth is necessary, but the extremities must be exposed to the air under dry coolness and the feet should be elevated on pillows Bed rest and nutritional support assist the local condition Amputation is rarely necessary

FOREIGN BODIES AND GUNSHOT WOUNDS

FOREIGN BODIES

Foreign bodies may produce acute symptoms or they may remain asymptomatic notwithstanding their presence in the tissues for a long time

Fig 68 —Roentgenogram of foot taken for other purposes disclosed needle Asymptomatic

Needles are the commonest type of foreign body to penetrate the deep structures of the foot Most patients are seen soon after the accident, because of disabling pain, however, I have seen twelve asymptomatic cases disclosed when the patients were examined and treated for another purpose (Figs 68 and 69) A needle's presence in the tissues sometimes becomes symptomatic after lying dormant for years, especially if the needle is embedded in the plantar

Fig 69—Part of needle in great toe Presence of foreign body dormant for a long time, suddenly became symptomatic

Fig 70—Part of needle in foot Acute symptoms immediately after entrance

Fig 71 —Small piece of glass embedded in subcutaneous tissue of plantar surface Painful

Fig 72 —Sharp piece of glass embedded between the fourth and fifth metatarsal heads Extensive osseous proliferation

surface (Fig 70) Long-standing asymptomatic needles do not call for removal A needle that causes pain should be excised The needle should be carefully located in the foot roentgenographically before operation is undertaken Unless the needle is close to the surface, its removal may be difficult

Small pieces of *glass* occasionally penetrate below the skin and become encapsulated (Fig 71), they remain relatively superficial and are readily excised Larger fragments may become wedged in an intermetatarsal space, when they are not removed, necrosis results (Fig 72)

Exogenous hair in the sole of the foot may become painful While walking barefooted a person may pick up a hair without its being noticed, it then becomes embedded in the epidermis of the sole of the foot, where it remains invisible and inert At a later date, the hair may set up an acute inflammatory process, but the cause at first is obscure Sometimes the area must be probed under magnification until the hair is found, sometimes when the area suppurates, the hair may be exuded with the suppurative material

Fig 73 —Fragments of gunshot Constant pain in the tarsal joint

GUNSHOT WOUNDS

The emergency treatment of gunshot wounds immediately after occurrence is not within the scope of this book Gunshot wounds may leave deformities of the foot, requiring surgical intervention, but they vary so much that a single method cannot be outlined Each case must be studied for correction of the particular deformity

Remaining particles of gunshot (Fig 73) often produce delayed symptoms of varying extensiveness Only pain-producing particles require excision Roentgenographic preparatory pinpointing of the site of the particles in the foot is always necessary

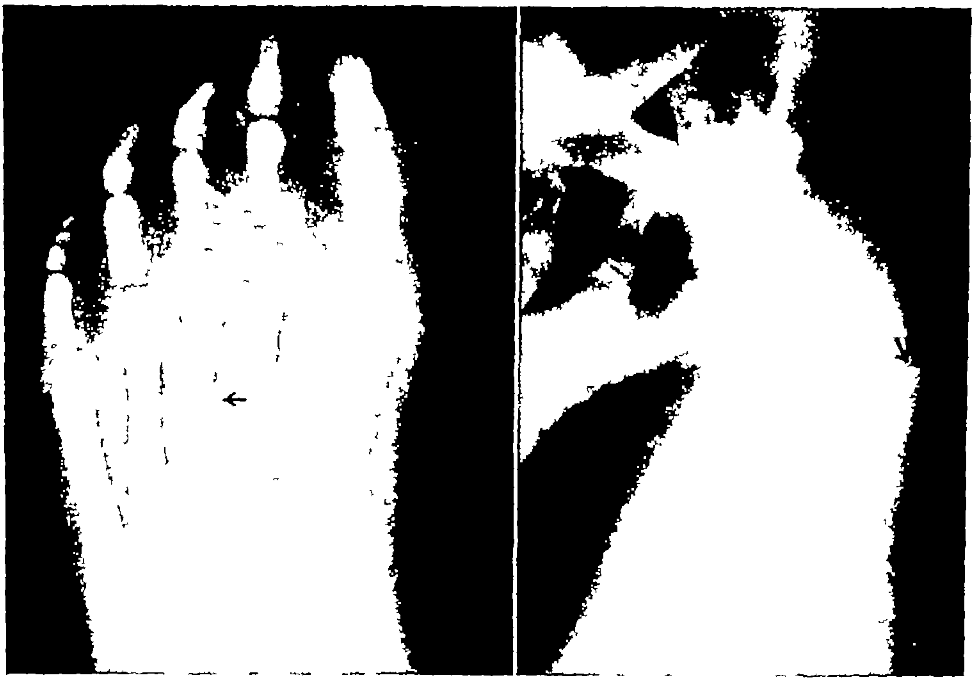


Fig 71 —Small piece of glass embedded in subcutaneous tissue of plantar surface Painful

Fig 72 —Sharp piece of glass embedded between the fourth and fifth metatarsal heads Extensive osseous proliferation

pen when walking or running, the line of force goes through the ankle laterally or medially to the body of the talus At that moment, weight is not transferred through the foot, the line of force escapes through the side of the ankle, thus spraining or lacerating the ligament which is not capable of absorbing the force.

Kelly (1952) is of the opinion that common ankle injuries represent the result of an exaggeration of one basic foot motion, either inversion or eversion Galland (1940) believes that in treating ankle sprain, attention should be focused on the talocalcaneal articulation rather than on the ankle mortise The motions of the ankle joint are almost entirely those of dorsiflexion and plantar flexion, motions not commonly made in incurring the average sprain, the motions involved are adduction and abduction, movements that take place particularly in the subtalar articulations and consequently affect the calcaneum That is why Galland adapted the political saying to this purpose· "As the calcaneum goes, so goes the foot"

It is my belief that it is usually an adduction and inversion which causes the common sprain on the lateral side An abduction and eversion causes the uncommon sprain on the medial side of the ankle

The frequent observation that a severe ankle sprain is worse than a fractured ankle is true A fracture is readily recognized so that appropriate treatment may be instituted at once, whereas sprains are often regarded lightly except in rare cases in which dislocation of the ankle is evident Collateral ligaments may be completely ruptured in ankle sprains, and there may be an associated diastasis of the tibiofibular ligaments with a dislocation of the ankle mortise. Such sprains are usually reduced spontaneously by muscle spasm Occasionally manual correction is done Sprains that reduce spontaneously are often neglected and not treated by complete immobilization or repair of the ligaments The resulting unstable ankle may require major surgical repair at a later date

Injuries to Lateral Malleolar Ligaments

Injuries to the lateral malleolar ligaments have three classifications (1) sprains of the lateral collateral ligaments, (2) avulsion and momentary dislocation of the ankle joint, and (3) recurrent dislocations

Simple Sprains.—Recovery from simple sprains is complete in a few weeks, nevertheless, every sprained ankle should be investigated to make certain that the sprain is actually simple

Moderate or Severe Sprain.—Most ankle injuries are moderate or severe sprains Injury to the ligaments varies from extreme stretching of the collateral ligaments to tearing of the ligaments without complete avulsion

Symptoms—Swelling and local tenderness over the sinus tarsi, painful weight bearing, and increased discomfort on forced inversion of the foot comprise the symptoms Edema is partly due to subdermal bleeding, and ecchymoses is an accompanying manifestation of subdermal bleeding

Diagnosis—If complete avulsion of the lateral collateral ligaments has occurred, placing the foot in an inverted position may demonstrate displacement

SPRAINS AND ATHLETIC INJURIES

A *sprain* is a wrenched or twisted joint with partial rupture or complete tearing of its attachments, which are mainly ligaments. Ligaments are tough, flexible nonelastic bands of tissue which attach to joint ends of bones and may cover the joints themselves. A *strain* describes stretched or overused muscles, tendons, or ligaments, without a tear in any of those structures. Sudden injuries of the foot may be grouped into those due to violent accidents and those occurring during normal use of the foot. Sudden injuries due to accidents include any degree of crushing, tearing, or breaking of one structure or a group of components of the foot and ankle, whereas sudden injuries that take place during normal use for the most part involve the ligaments. When they are accompanied by fractures, as they are only occasionally, the fracture is an infraction or chip fracture.

A *sprain* is caused by sudden twisting, or torsion, of a joint beyond its limitation or motion; in other words, extraordinary stress is exerted on the ligaments of that joint. The foot and ankle are more subject to sprains than any other part of the body. Sprains vary from a mild stretching of the fascial components to complete laceration of the ligaments and capsular composition. The lateral aspect of the ankle is by far the site sprained most; at times, the medial aspect is sprained.

Structure

The distal ends of the tibia and fibula form a mortise which maintains the trochlear surface of the talus (Fig. 8), allowing a hinge motion in this joint. The anterior portion of the trochlear surface of the talus is wider than the posterior. It is held in the mortise on the medial aspect by the medial malleolus and on the lateral, by the lower end of the fibula. The greater width of the anterior part of the body of the talus places the fibula in slightly external rotation. The ligaments about the ankle joint maintain the anatomic mortise. There are three groups of ligaments: (1) the medial collateral or deltoid ligaments, (2) the lateral collateral ligaments, and (3) the inferior tibiofibular ligaments, also called the tibiofibular syndesmosis.

The *medial collateral ligament (deltoid ligament)* originates from the entire border of the medial malleolus, extending anteriorly into most of the medial aspect of the talus and into the sustentaculum tali, and some filaments into the navicular. The *lateral collateral ligament* is composed of three separate and distinct fascial bands: the posterior talofibular ligament, the anterior talofibular ligament, and the calcaneofibular ligament; these are the collateral ligaments commonly affected in ankle sprain. The inferior tibiofibular ligaments (the syndesmosis) bind the malleoli to hold the talus in mortise.

Mechanism

Normally, the thrust of the body weight in motion is transmitted through the leg to the talus and then through the foot to the supporting surface. When the foot is turned suddenly in extreme abduction or adduction, which may hap-

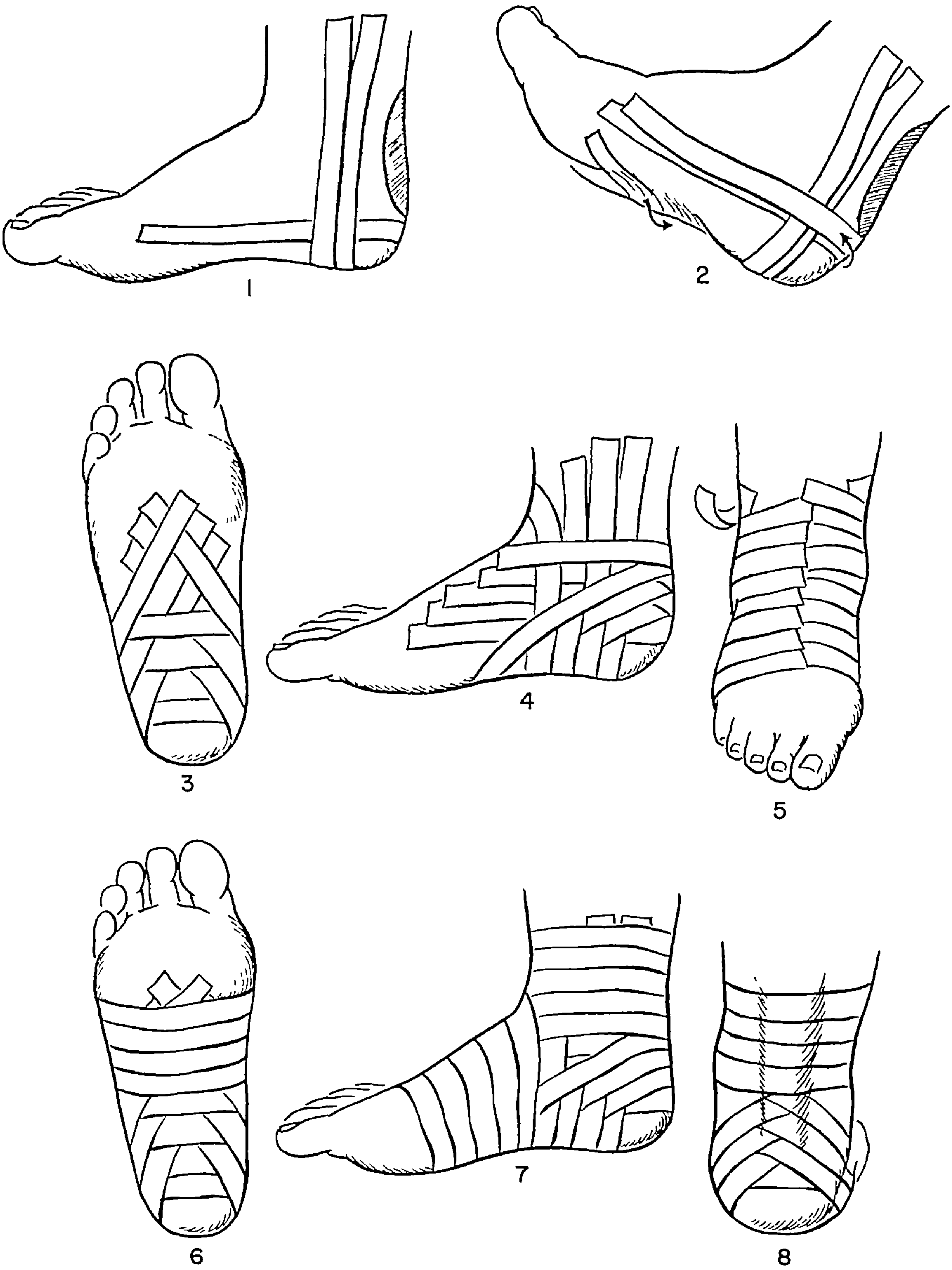


Fig 75 —Immobilization of ankle joint with adhesive by Galland's method

of the head of the talus in front of the lateral malleolus When the patient is seen immediately or within a few hours after injury, precise diagnosis is often difficult

Treatment—The treatment for contusions, sprains, and strains is as follows

1 Immediate treatment consists of (a) cold applications to constrict the arterial beds and reduce subdermal bleeding, (b) compression to arrest active subdermal bleeding, (c) elevation of the injured part to decrease edema by simple hydrostatics, and (d) rest, which is in any case imposed by pain as a matter of course Obviously, excessive manipulation of the foot aggravates a recent injury and consequently lengthens the period of disability Cold applications begin to lose their value after about twenty-four hours, whereas compression and

Fig 74—Severe inversion and subluxation of ankle joint result of complete rupture of lateral malleolar ligaments (From Lee, H G J Bone & Joint Surg 39A 828-833, July, 1957)

elevation may be helpful for several days Moreover, cold and compression are used guardedly, if at all, for aged patients or patients with peripheral vascular disease

2 After removal of the compression dressing the next day, assess the extent of the injury By palpation and gentle manipulation, ascertain whether there has been a simple stretching or complete avulsion of the ligaments Such grading is prognostically significant When the degree of injury is uncertain, roentgenograms should be taken under anesthesia, with the ankle stressed in eversion and inversion so as to rule out complete rupture of the ligaments (Fig 74)

3 In about thirty-six hours when the danger of further subdermal bleeding passes, if edema is minimal, apply heat rather than cold to dilate peripheral vessels, thus speeding absorption at the site of injury

procedure is best done immediately after the injury, particularly when there are abrasions, which may become the source of sepsis; otherwise, operation is preferably deferred a few days until the patient has recovered from the immediate effects of injury.

Anderson and Lecocq (1954) reported twenty-seven cases of repair of collateral fibular ligaments. They observed that many so-called minor ankle sprains are in reality complete ruptures of the collateral malleolar ligaments and that early repair is unqualifiedly indicated in most of those cases. Those authors are in accord with Leonard (1949) regarding the primary importance of reconstructing the anterior talofibular ligament in the corrective procedure, differing with

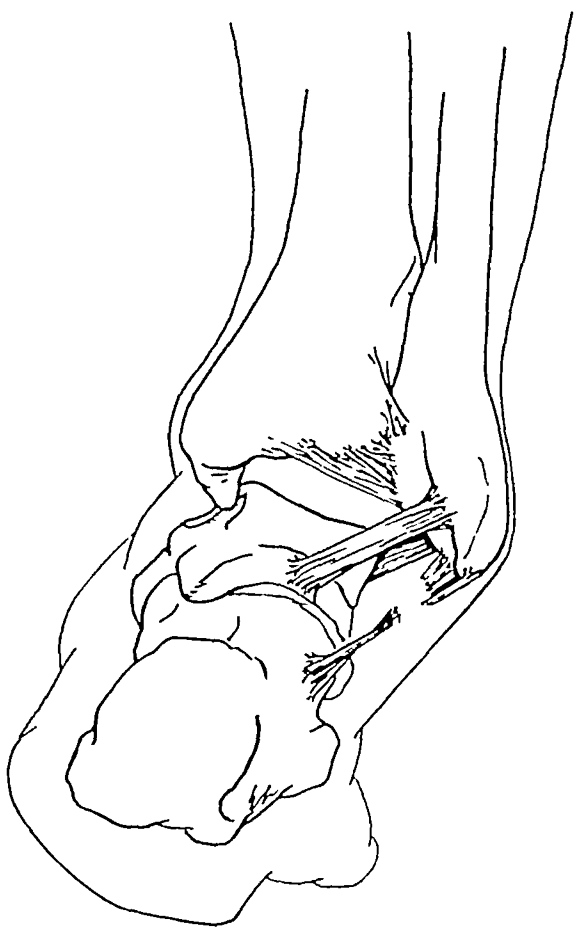


Fig 76 —Rupture of lateral malleolar and inferior tibiofibular ligaments

Bonnin (1944), who believes that the calcaneofibular ligament is of first importance and that its stability may be restored by the tenodesis of Watson-Jones (1952), in which the peroneus tendon is utilized as replacement tissue I am in agreement with Bonnin (1944) to the extent that the calcaneofibular ligament is at least as important as the anterior talofibular ligament Watson-Jones' tenodesis, however, is not applicable in cases of proved avulsion of the collateral ligaments occurring shortly after an accident In such cases any torn ligaments are repaired at operation In no two cases will the tear be of the same ligaments Watson-Jones' tenodesis is of value only in old recurrent sprained ankles In acute cases in which repair is attempted shortly after the injury, if coaptation and

4 In general, when swelling is moderate and complete rupture of the collateral ligament has been ruled out, partial immobilization with adhesive tape or a firm ankle support, with or without the injection of procaine hydrochloride, permits healing while the patient is ambulatory Galland (1940) gives a comprehensive, detailed, and illustrated description of the method of strapping a sprained ankle (Fig 75) Briefly, the strapping is applied at appropriate inclinations of the foot in rotating successions of an average of three sets of straps, one longitudinal and two transverse The straps are applied in an interlocking manner to cover the sole and sides of the foot beneath a completely encircling strapping of the foot and ankle Galland acknowledges the danger of circulatory compression unless tension of the straps is uniform throughout the dressing

Whenever there is any doubt about the severity of an injury, it is safer to apply a plaster walking cast for three to five weeks

Infiltration of 5 to 10 ml of 1 per cent procaine hydrochloride has been advocated by many for sprained ankle without avulsion of the ligaments McMaster (1943) reported 400 cases of sprained ankle in which 200 were treated by injection of procaine hydrochloride, which permitted immediate normal use of the ankle, and 200 were treated by immobilization with adhesive strapping He reports that treatment by injection of procaine hydrochloride alone uniformly gave the better results It is difficult to reconcile the continued normal use of injured tissue without interference with its healing process

Avulsion of Collateral Ligaments

Avulsion of the collateral ligaments is the severest of all injuries to the lateral side of the ankle In most instances the collateral ligaments are completely torn, so that a flail ankle joint and a diastasis of the tibiofibular syndesmosis (Fig 76) complicate the injury Leonard (1949), who experimented with seven preserved ankles and studied fifty-one sprained ankles, concluded that the most important component of the collateral group in avulsion is the anterior talofibular ligament In my opinion the calcaneofibular ligament is equally important

Symptoms differ only in degree from those in the ordinary sprained ankle Special roentgenograms made while the ankle is forcefully inverted are indicated when a sprained ankle is extremely swollen and painful Infiltration of 10 ml of 1 per cent procaine hydrochloride into the area of maximum tenderness permits placing the foot in extreme inversion Clayton and his associates (1951) recommend blocking the peroneal nerve by injection of 5 to 10 ml of 2 per cent procaine hydrochloride around the peroneal nerve as it winds around the neck of the fibula I recommend instead a light Pentothal sodium anesthetic

Immediate treatment is the same as that described for the simple sprained ankle In a day or two when edema has stabilized, complete immobilization in a walking cast is instituted and maintained for six to eight weeks After removal of the cast, massage, passive exercises, and an elastic ankle support are prescribed Carefully fitted broad-heeled shoes must be worn for four to six months

In serious and complicated cases, open reconstruction and restoration of normal continuity of the torn structures are advisable Sometimes operative

stabilizes the ankle sufficiently to prevent further sprains Some patients may be helped by a short leg brace with a T strap to prevent inversion

Surgical Treatment.—In intractable cases, *Watson-Jones' tenodesis* (1952) is the most efficient method of stabilizing the ankle Kelley and Janes (1956) reported seven cases of recurrent sprained ankle in patients ranging in age from 18 to 56 years, whose average duration of symptoms had been seven years Peroneal brevis tenodesis was performed with excellent results When the ligamentous tissue is frayed so that repair is impossible, the Watson-Jones tenodesis is satisfactory, provided the hole is not drilled into the neck of the talus Watson-Jones himself recognized that drilling the hole in the neck of the talus was perhaps an unnecessary step

In a personal series of seventy-five cases, I either repaired the old tear and transferred over it a piece of fascia lata or deliberately made two incisions in the ligaments and then sutured them This is comparable to my procedure for repairing the deltoid ligament (Fig 80)

Watson-Jones' Tenodesis.—For instability of the lateral aspect of the ankle, Watson-Jones' tenodesis is applicable

1 Make a hockey-stick incision, beginning behind the lower third of the shaft of the fibula, extending downward, and rounding an inch below the tip of the lateral malleolus to the calcaneo-cuboid junction

2 Divide and retract the deep fascia

3 Dissect the peroneus brevis tendon from its muscle and suture the muscle fibers to the tendon of peroneus longus

4 Free the tendon as far as the lateral malleolus Do not disturb the annular fibers that hold the tendon in position behind this bone

5 Drill a hole, ¼ inch in diameter, horizontally from the posterior or to the anterior margin of the malleolus, drill a second similar hole ½ inch above the first and a third hole vertically in the outer margin of the neck of the talus adjacent to the articular surface, emerging in the roof of the sinus tarsi

6 Guide the tendon through the superior drill hole in the malleolus, through the drill hole in the talus, and then through the inferior hole in the malleolus There, suture the tendon to itself and to the posterior band of the lateral ligament

Watson-Jones' procedure does present difficulties, especially in making the drill hole in the neck of the talus and guiding the tendon through that hole, consequently, several modifications have been suggested

Lee's Modified Tenodesis.—Lee's modification (1957) is simple and less traumatic and was equally as satisfactory as the original Watson-Jones' tenodesis in his seven cases of extreme instability of the lateral aspect of the ankle He followed his patients for twelve years At this writing, I have not employed Lee's rational procedure, but it is my intention to do so

1 Make a hockey-stick incision, beginning behind the lower third of the fibula, coursing downward and around the lateral malleolus, and ending at the calcaneo-cuboid joint

repair of the ligaments cannot be accomplished, a fascia lata graft may be transferred over the ligaments and sutured to them

Recurrent Sprained Ankles

Recurrent dislocation of the ankle, weak ankles, and *chronic sprains* are terms loosely applied to injured ankle ligaments Recurrent episodes of overstretching and spraining of ankle ligaments ultimately leave them completely stretched or torn This eventuality is more likely in persons who have congenitally weak or flaccid ligaments The condition, however, may be due to sudden avulsion of the collateral ligaments with spontaneous reduction of the talus dislocation, which, if not properly treated, results in an unstable ankle. A sprained ankle may and often does lead to permanent disability (Bonnin, 1944) In a

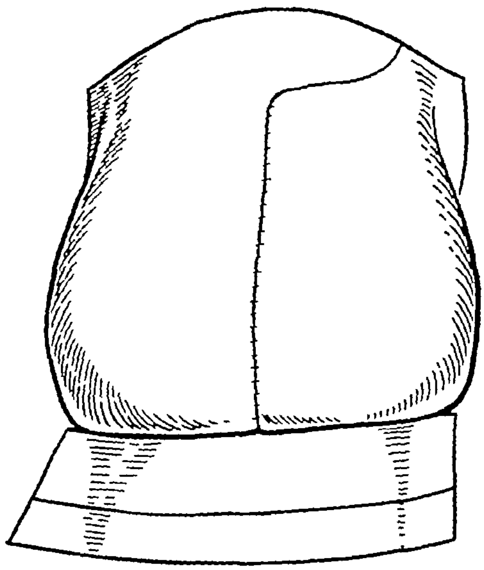


Fig 77 —Outflared lateral border of heel on boot helps prevent recurrent sprained ankle

report of fifty-seven cases of injury to the ankle without bony changes, Hughes (1942) recorded some lateral tilting of the ankle in about half the cases when roentgenograms were made with the ankle held in forced inversion Bosien and his co-workers (1955) followed 133 cases of ankle sprains for an average of twenty-seven months Among their patients, 36 per cent had had previous injury to the same ankle and 33 per cent had had continuous residual ankle symptoms

Repeated twisting or giving-way of the ankle is typical, especially when walking on irregular ground and when wearing heels worn down on the outer posterior surface Among women, recurrent ankle sprain often results from previous injuries to the ankle coupled with wearing high-heeled shoes The base of such heels is sometimes less than 2 cm in diameter, and such a shoe often does not have a counter to support the ankle, consequently, ankle sprains are inevitable

Supportive Treatment.—A good supportive shoe, with an outflared broad-based heel (Fig 77), in addition to an elastic ankle support, in some cases

2 Incise the fascia, exposing the peroneal tendons

3 Strip the peroneus brevis from its muscle belly as high as possible to obtain sufficient length to form a long loop through the malleolus (Fig 78, *A*).

4. Suture the loose muscle fibers of the peroneus brevis to the aponeurosis of the peroneus longus.

5 Free the peroneus brevis tendon down to the superior peroneal retinaculum, taking care not to disturb the ligamentous fibers

6 Drill a horizontal hole, ¼ inch in diameter, just below the broadest part of the lateral malleolus

7 Thread the peroneus brevis tendon through the hole from back to front while the foot is held everted in correct valgus position and the tendon is pulled downward below the lateral malleolus There, suture the tendon to itself and to the sheath of the peroneus longus

8 Denude a flap of fascia from the lower portions of the fibula and tibia, fold the flap downward, and suture it to the surrounding tissues (Fig 78, *D*)

9 Close the wound in layers, with the foot held in mild valgus and eversion

10 Apply a plaster splint

In about ten days remove the sutures, apply a walking cast, and permit weight bearing On removal of the cast six weeks later, the patient wears, for about six months, an elastic ankle support and a well-fitted Oxford or high shoe with an outflared heel

Injuries to Medial Ligaments of the Ankle

The medial side of the ankle is not sprained often When it is, it is caused mostly by sudden eversion and abduction of the foot which subjects the deltoid ligament to extreme stress The injury may tear the deltoid ligament, when it does, the laceration is likely to be accompanied by fracture of the tip of the medial malleolus

Conservative Treatment.—Treatment is according to the degree of injury In simple sprains, when the deltoid ligament has not been torn, partial immobilization with adhesive or an ankle support for three weeks is sufficient for recovery All cases of avulsion of the deltoid ligament should be considered serious, requiring complete immobilization by plaster cast Dziob (1956) suggested that in treating medial avulsion of the ankle, when the tilt of the talus in inversion under stress roentgenography is less than 15 degrees, plaster immobilization of the foot and leg for six to eight weeks is adequate, however, when greater than 15 degrees, the torn ligaments must be sutured

Surgical Treatment.—The Schoolfield and DuVries techniques follow

Schoolfield's Technique—Schoolfield's technique was originally intended for flatfoot but has been adapted to recurrent sprains of the deltoid ligament A semilunar incision is made, beginning behind the medial malleolus, coursing downward and curving anteriorly about 2 cm below the malleolus, and ending over the tuberosity of the navicular The anterior skin flap is denuded and deflected to expose the deltoid ligament which is detached from the medial malleolus and freed to its insertions The foot is inverted at this point to permit

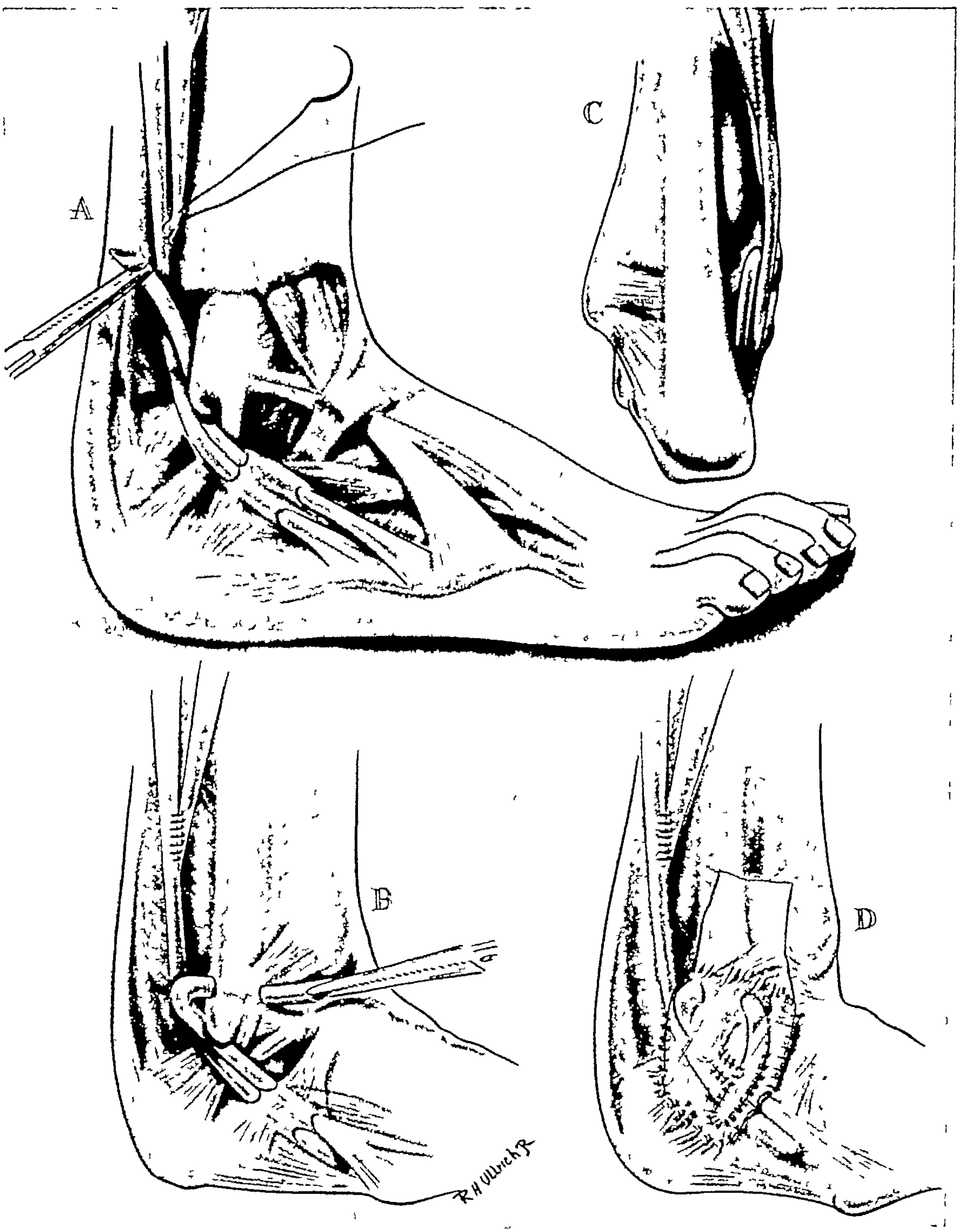


Fig 78 —Lee's tenodesis for stabilization of ankle *A*, Stripping of peroneus brevis tendon *B*, Sutured proximal end of peroneus brevis to peroneus longus, distal end of peroneus brevis tendon guided through drill hole in lateral malleolus *C*, Posterior view *D*, Distal end of peroneus brevis tendon sutured to itself and to peroneus longus sheath, covered with flap of fascia stripped from malleolus (From Lee, H. G J Bone & Joint Surg 39A 828-833, July, 1957)

DuVries' Technique—For recurrent sprains of the deltoid ligament, Schoolfield's operation (1952) for shortening of the deltoid ligament is effective in repairing and reinforcing the medial aspect of the ankle, however, the crucial incision and cross-shaped scar formations give preferable results

Bisect a vertical incision by a horizontal incision, forming a cross of equal arms Suture the margins together to leave a cross-shaped scar which is strong enough to act successfully as a stabilizer of the ankle (Fig 80)

Sprains of First Metatarsophalangeal Joint and of Plantar Fascia

First Metatarsophalangeal Joint.—The first metatarsophalangeal joint is sometimes sprained by stubbing the toe or by extreme dorsiflexion Swelling over the joint and pain on motion are symptomatic. Such sprains are for the most part simple sprains and respond to partial immobilization with adhesive tape and the wearing of a shoe with a rigid sole

Plantar Fascia.—The plantar fascia may be sprained by jumping, especially from heights The sprain may be anywhere along the length of the fascia but oftenest at its origin Sprains of the plantar fascia respond to strapping and the wearing of a shoe with a rigid shank and a longitudinal arch support

Athletic Injuries

The prevention and treatment of athletic injuries are the concern of the physician who must have the cooperation of all personnel directly associated with the athletic activity, whether of a school or club Novich (1956) has explained the responsibilities of the team physician in a thorough article in which he stresses the importance of the medical history of every player and of a physical examination before each game He thinks rightly that the team physician should be on hand at hazardous athletic events and that treatment of injuries should include follow-up after the season The coaches and sponsors should cooperate in seeing that athletes undergo appropriate conditioning and adequate long-term care

The foot and ankle are commonly traumatized during athletics, a sprained ankle is always a threat in sports, such as baseball, football, and hockey Thorndike (1948), in an analysis of athletic injuries on the Harvard football team from 1932 to 1947, found 585 ankle sprains and only 15 fractured ankles

Other common athletic injuries are contusion, sprains in the forefoot, especially the metatarsophalangeal joints, fractures of the phalanges, usually of the fifth or of the hallux, and traumatic tenosynovitis Treatment for specific injuries are discussed under their appropriate headings elsewhere in this volume

Preventive treatment is essential Athletes who are exposed to violent twisting of the ankle should have their ankles strapped with adhesive tape so as to limit inversion and eversion to a minimum A nonelastic ankle support can be worn during games This applies especially to athletes who have had recurrent ankle sprains For protection of the forefoot, shoes should have rigid or metal toe boxing and firm soles.

suturing the deltoid ligament to the periosteal and ligamentous tissues over the medial malleolus just above the natural origin of the deltoid ligament (Fig 79). (This takes up the slack in the deltoid ligament) A walking cast is applied with the foot held in mild inversion for six weeks

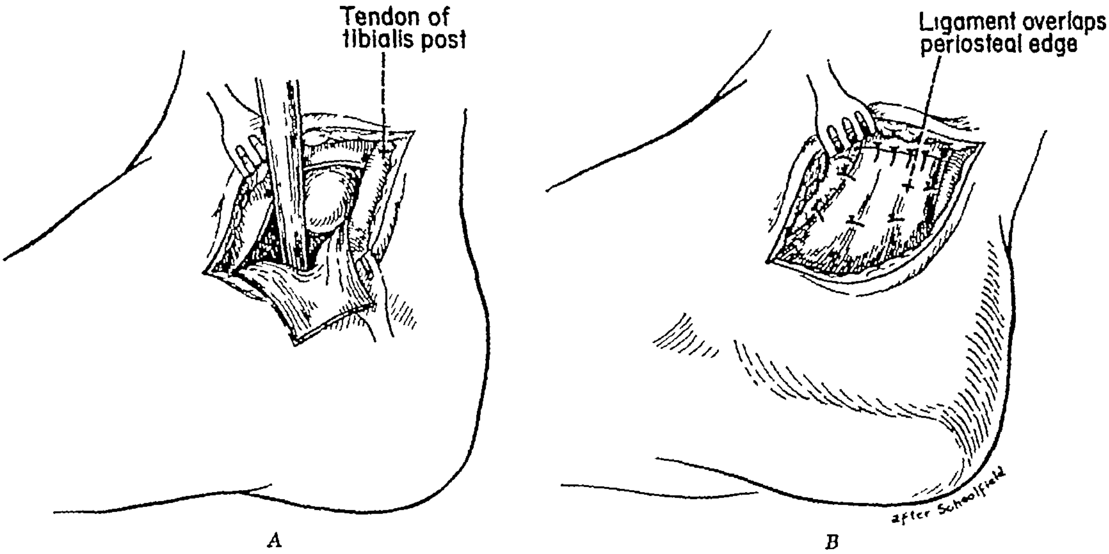


Fig 79 —Schoolfield's technique *A*, Deltoid ligament stripped from above downward over medial malleolus *B*, Foot held in inversion, upper margin of deltoid ligament is sutured to periosteal edge which it overlaps

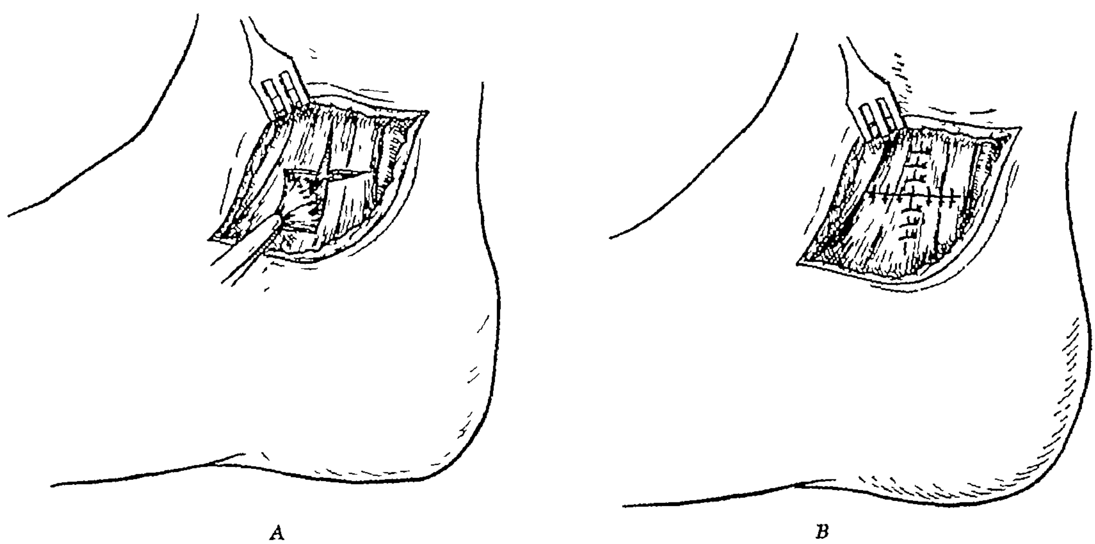


Fig 80 —DuVries' technique Crucial incision *A*, Vertical incision into deltoid ligament bisected at center by a transverse incision, four right-angular flaps of ligament resulting are denuded and freed from bone *B*, Four flaps sutured to form a cross-shaped scar which stabilizes ankle

CHAPTER SIX

Ulcers; The Foot in Diabetes

ULCERS

An ulcer is usually a single symptom in a disease syndrome The lower extremities are highly susceptible to ulceration Ulcers resulting from varicosities, diabetes, and arteriosclerosis are characteristically chronic The particular types of ulcers often encountered on the foot are (1) traumatic, (2) perforating, (3) trophic, (4) necrotic, (5) varicose or indolent, (6) rodent or malignant, and (7) diabetic

Traumatic Ulcer

Traumatic ulcers are due to excessive pressure on a weight-bearing area over a long period Degeneration of the skin surface and underlying soft structures leads to ulceration Ulcers are commonest under the metatarsal heads but may appear over any bony prominence (Figs 81 and 82) They are seen often in older persons, because of reduced general tissue vitality and long-standing irritation General diseases, such as diabetes and disorders producing anemia, accelerate the process

The ulcer is characterized by large amounts of granulation tissue surrounding a small orifice which exudes a nonpurulent discharge Ulcers are subject to recurrent secondary infection which may become intractable

In treatment, (1) excise all surrounding overlying callus, (2) cauterize the granulation tissue with 90 per cent phenol or 10 per cent silver nitrate, (3) paint the ulcer with a dye, such as carbolfuchsin or gentian violet, (4) protect the area from further pressure by padding or a foot appliance

Traumatic ulcers may have chemical and thermal causation, in which case treatment is the same as for chemical or thermal burns.

REFERENCES

Burns and Freezing

Blair, J R , Schatzki, Richard, and Orr, K D Sequelae to Cold Injury in One Hundred Patients, J A M A **163** 1203-1208, April 6, 1957

Bigelow, W G The Modern Conception and Treatment of Frostbite, Canad M A J **47** 529-534, Dec , 1942

Brownrigg, G M Frostbite in Shipwrecked Mariners, Am J Surg **59** 232-247, Feb , 1943

Edwards, E A , and Leeper, R W Frostbite An Analysis of 71 Cases, J A M A **149** 1199-1205, July 26, 1952

Fausel, E G , and Hemphill, J A Study of Late Symptoms of Cases of Immersion Foot, Surg Gynec & Obst **81** 500-503, Nov , 1945

Kaye, B B Burns An Outline for Treatment, Am J Surg **92** 123-138, July, 1956

Lewis, T Observations on Some Normal and Injurious Effects of Cold Upon the Skin and Underlying Tissues, Frostbite (Holme Lecture), Brit M J **2** 869-871, Dec 20, 1941

Ungley, C C Treatment of Immersion Foot by Dry Cooling, Lancet **1** 681-682, May 29, 1943

Vinson, H A , and Schatzki, Richard Roentgenologic Bone Changes Encountered in Frostbite, Korea 1950-51, Radiology **63** 685-695, Nov , 1954

Webster, D R , Woolhouse, F M , and Johnston, J L Immersion Foot, J Bone & Joint Surg **24** 785-794, Oct , 1942

Sprains and Athletic Injuries

Anderson, K J , and Lecocq, J F Operative Treatment of Injury to the Fibular Collateral Ligament of the Ankle, J Bone & Joint Surg **36A** 825-832, July, 1954

Bonnin, J G The Hypermobile Ankle, Proc Roy Soc Med **37** 282-286, Jan , 1944

Bosien, W R , Staples, O S , and Russell, S W Residual Disability Following Acute Ankle Sprains, J Bone & Joint Surg **37A** 1237-1243, Dec , 1955

Clayton, M L , Trott, A W , and Ulin, Robert Recurrent Subluxation of the Ankle, With Special Reference to Peroneal Nerve Block as a Diagnostic Aid, J Bone & Joint Surg **33A** 502-504, April, 1951

Dziob, J M Ligamentous Injuries About the Ankle Joint, Am J Surg **91** 692, April, 1956

Galland, W I An Adhesive Strapping for Sprain of the Ankle, J Bone & Joint Surg **22** 211-215, Jan , 1940

Hughes, J R Sprains and Subluxations of Ankle-Joint, Proc Roy Soc Med **35** 765-766, Oct , 1942

Kelley, J H , and Janes, J M The Chronic Subluxating Ankle, A M A Arch Surg **72** 618-621, April, 1956

Kelly, R P Ankle Injuries, Kentucky M J **50** 281-288, 1952

Lee, H G Surgical Repair in Recurrent Dislocation of the Ankle Joint, J Bone & Joint Surg **39A** 828-833, July, 1957

Leonard, M H Injuries of the Lateral Ligaments of the Ankle, A Clinical and Experimental Study, J Bone & Joint Surg **31A** 373-377, April, 1949

McMaster, P E Treatment of Ankle Sprain, Observations in More Than 500 Cases, J A M A **122** 659-660, July 3, 1943

Novich, M M A Physician Looks at Athletics, J A M A **161** 573-576, June 16, 1956

Schoolfield, B L Operative Treatment of Flatfoot, Surg Gynec & Obst **94** 136-140, Feb , 1952

Thorndike, A Athletic Injuries, Prevention, Diagnosis, and Treatment, Philadelphia, 1948, Lea & Febiger

Watson-Jones, Reginald Fractures and Joint Injuries, ed 4, Baltimore, 1952, Williams & Wilkins Co , vol II, pp 821-823

Fig 82 – Traumatic ulcer over second toe Acute tinea infection of all toes (Courtesy Dr Ned Pickett)

Fig 83 – Perforating ulcer under first metatarsal head

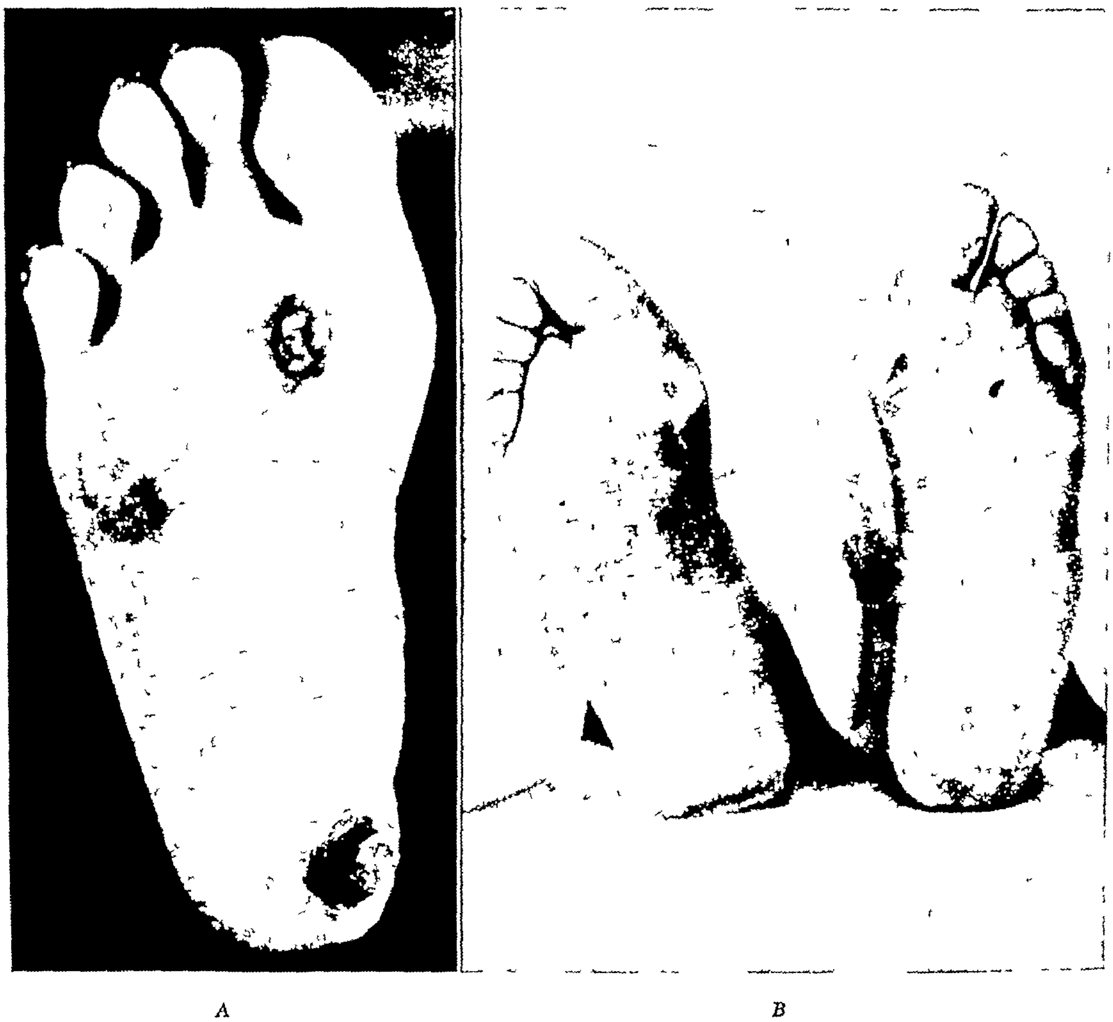


Fig 81 — A, Traumatic ulcers under second and fifth metatarsal heads and under heel B, Traumatic (pressure) ulcers Right foot first metatarsal head, left foot first and third metatarsal heads

Perforating Ulcer

Perforating ulcer represents a low-grade degeneration, usually on the sole of the foot It is frequently under the great toe joint where it forms an opening on the dorsum of the foot, hence, it is called *perforating* (Fig 83) From its orifice, it may penetrate into a joint or into caries of bone

Alcoholism, syphilis, diabetes, arteriosclerosis, and peripheral nerve disease predispose to perforating ulcers Pressure on a corn or callus, long continued, especially over a bony prominence, and abrasion are exciting factors Tocantins and Reimann (1939) reported a familial occurrence of trophic changes and vasomotor disturbances as well as dissociated sensory changes of the extremities The disorder was first exhibited as perforating ulcers of the feet, which seemed attributable to an organized dysgenesis of the central nervous system Smith (1934) also reported on familial neurotrophic osseous atrophy of the feet, in which leprosy was suggested Kulowski and Perlman (1936) believed that all "osteomye-

The primary objective in treating varicose ulcers is occlusion of the varices proximal to and under the ulcer This may be accomplished by one of the following procedures, utilizing any combination of appropriate steps.

1 A sponge-rubber compression bandage, sometimes called *venous heart*, may be used A nonirritating medication, such as a dye or an antibiotic ointment, is applied to the ulcer and covered with sterile gauze. A sponge-rubber pad, about ½ inch thick, is placed over the gauze, covering the entire ulcer and extending about 3 cm proximally to the ulcer, then an elastic bandage is wrapped around the foot and leg

2 A second method of treatment is to eliminate the varices that caused the ulcer by sclerosing, ligating, or stripping the varicosed vein Supportive measures, such as wearing elastic stockings or bandages, should be used after the ulcer has healed so as to prevent recurrence

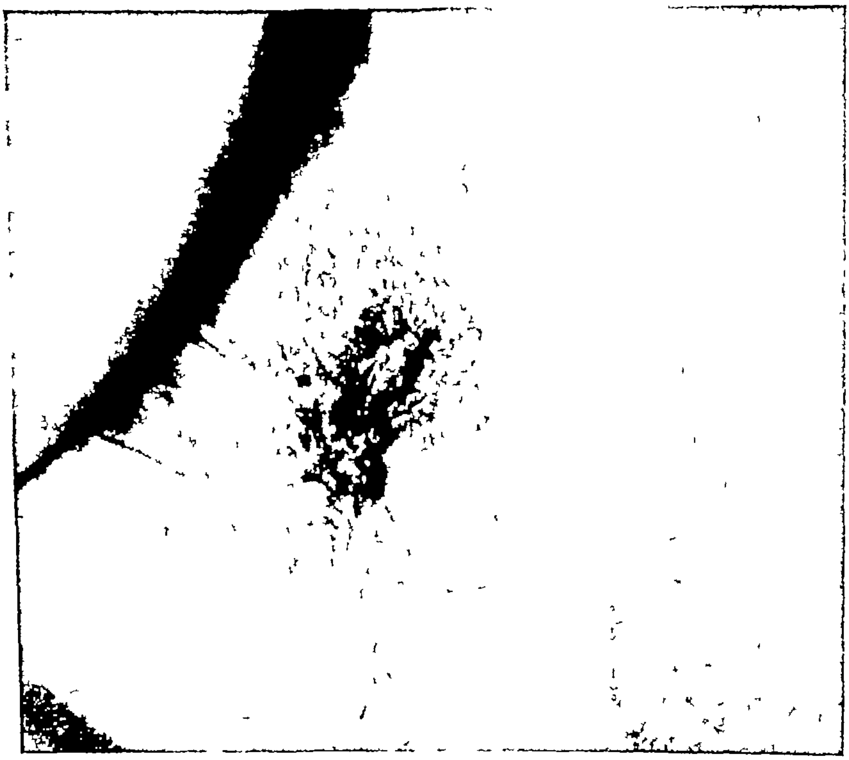


Fig 84 —Varicose ulcer over medial malleolus (Courtesy Dr Ned Pickett)

Malignant Ulcer or Rodent Ulcer

The malignant or rodent ulcer is a slowly growing variety of squamous cell carcinoma (basal cell carcinoma) The skin and subcutaneous tissues of the area slowly erode The base of the area is adherent to the surrounding tissues It resists all forms of medicinal therapy If the diagnosis is confirmed by biopsy, wide excision of the growth is indicated It rarely occurs on the foot

Diabetic Ulcer of the Foot

Ulcer of the foot is a common complication of diabetes The ulcer appears on the plantar surface, especially under a weight-bearing pivot The plantar

litic foci in perforating ulcer of the foot should be radically" treated, but that amputation should be a last resort because "ulceration has been known to recur in the stump and also in the other foot '

In treatment for perforating ulcer, remove the cause whenever possible, rest the area as much as possible With caution, curet away all degenerated tissue Apply mild heat Use foot appliances, padding, or shoe wedging to alter weight bearing

Trophic Ulcer

Trophic ulcers are always due to degenerative changes of the peripheral nerves, usually of the roots of the lumbosacral spinal cord Formation of the ulcer is insidious The condition is accompanied by sensory changes, leading to anesthesia The patient, feeling no pain, may be unaware of the condition until there is complete breakdown of the tissues Trophic ulcer is likely to be secondary to such diseases as latent stages of pernicious anemia, spina bifida, syringomyelia, and syphilis of the spinal cord

Treatment is the same as for a perforating ulcer

Necrotic Ulcer

Necrotic ulcer is a slough of devitalized tissues such as takes place in diabetes, Buerger's disease, and arteriosclerosis and other vascular deficiencies The ulceration is often invaded by low-grade pathogens which cause the type of chronically sloughing, draining ulcer that forms on a weight-bearing surface

The first necessity in treatment is the control or amelioration of any underlying disease contributing to the formation of the ulcer Frequent débridement is essential, as is scrupulous cleanliness of the ulcerous area, which should be relieved of as much pressure as possible If the ulcer is on the plantar surface of the foot, weight bearing must be held to a minimum While weight bearing, heavy padding or an appliance must protect the area

Varicose Ulcer (Indolent Ulcer)

Congestive ulcers are due to a circulatory deficiency resulting in reduced tissue vitality Any interference in the blood supply to the foot may ultimately produce an ulcer, especially if the area is subjected to secondary irritating factors Varicose ulcer is the commonest congestive ulcer of the ankle It is sometimes called *indolent ulcer* It is a phlegmonous destruction of tissue caused by prolonged venous congestion or stagnated venous blood Such ulcers are secondary to long-standing varicose veins They occur just above the medial malleolus (Fig 84), rarely over other surfaces of the foot or ankle, they usually lie over a varix and are surrounded by varying degrees of pigmentation Because of prolonged and progressive deterioration of all the surrounding soft tissues, varicose ulcers are resistant to treatment They may hemorrhage spontaneously

THE FOOT IN DIABETES

There are two types of diabetic gangrene The first type occurs in diabetic patients who have advanced arteriosclerosis When gangrene sets in, it acts as it does in all occlusive vascular diseases in that it is rarely reversible and represents a terminal stage The second type of diabetic gangrene may take place in a warm foot in which the circulation is not seriously impaired and in which there is little if any evidence of arteriosclerosis It may even be possible to obtain a pulse This type ordinarily occurs in uncontrolled diabetes but may take place even when diabetes has been controlled The condition, however, is usually reversible, as it was in thirty personal cases When this type, sometimes called diabetic foot, threatens to become gangrenous, it can usually be successfully treated by conservative methods Only occasionally is amputation necessary, because of intractable pain or extensive tissue destruction Amputation is only justified under positive indications When amputation is inevitable, part of the foot can often be preserved for the patient to walk on, the preservation of any part of the natural foot prevents increased strain on the other foot, otherwise the excessive strain on the opposite member can result in similar complications, for the vessels in the opposite leg are likewise affected Loss of the diseased foot greatly increases the hazard of injuries to the remaining foot

Preventive Measures

Strict diabetic control is the most important single preventive measure Diabetic patients should guard against lacerations of the skin of their feet by keeping them protected from all possible injury Dryness and cracking can be counteracted by application of a bland skin oil Patients should be warned of the dangers of all forms of applied heat, strong antiseptics, and home remedies Hot-water bottles and electric pads are to be used only with utmost caution and under supervision, because sensation of pain and temperature is likely to be impaired Stockings should be large enough so as not to bind the toes and should be of thick material to provide some cushioning effect in the shoe Shoes should be preferably of an Oxford type, fitted to provide full freedom for the toes and prevent pressure or friction on any part of the foot

Toenails should be trimmed straight across and the corners rounded slightly with an emory board Corns and calluses should never be reduced by the patient or by untrained persons The callus on the sole of the foot with its devitalized subcutaneous tissue is a frequent site of chronic infection and deep ulcer That type of plantar ulcer is a constant invitation to organisms that can produce cellulitis of the foot The ulcerated corn and callus are often forerunners of gangrene Healing of the ulcer cannot be accomplished until the surrounding keratosis has been reduced and the area shielded by means of aperture pads to protect against further pressure

Fissures between the toes may result from a fungus infection or from excessive moisture They may also be the focus of infection and ulceration, which may be the inciting factor in diabetic gangrene Fissures may be prevented by watchful control

surface of the metatarsal heads, usually the middle three, is a likely area The ulcer is chronic and has a small orifice which exudes a seropurulent material The ulcer may penetrate the fascial layers The orifice is surrounded by a thick brawny white callus (Fig 85) On removal of the callus, the surface is seen to be covered with granulation tissue

First in importance in treatment is control of the underlying diabetes Cleanliness is imperative The brawny tissue surrounding the ulcer must be removed All secondary infections should receive careful attention The application of 5 per cent silver nitrate to the granulation tissue, repeated about twice a week, destroys the granulation tissue and permits healing by third intention All pressure should be removed from the area by means of padding or sponge-rubber shoe Local applications on large dressings, using a hydrophilic ointment containing water-soluble derivatives of chlorophyll (Chloresium), as described by

Fig 85 —Diabetic ulcer under fourth metatarsal head

Cady and Morgan (1948), is worth trying Cady and Morgan sought a healing preparation that would be applicable to all types of ulcers and effect early healing Milberg (1956) reported on the faster healing with softer scar and few recurrences obtained by Gelfoam in the treatment of chronic recalcitrant stasis of leg ulcers The product is a sterile powder, made from a gelatin, originally used for a physiologically absorbable sponge

Lucca (1956) reported on one hundred cases of leg ulcers, of which seventy-five were the chronic varicose type Results in his series were better when sympathectomy was performed before skin graft or when skin graft was not performed at all He recommended a one-stage operation for extirpation and skin grafting He reported 7 per cent recurrence after the skin graft in patients having other complications

If there is infection, antibiotic therapy must be instituted immediately. Because of impaired circulation, large doses of antibiotic, to which the sensitivity of the organism involved has been ascertained, should be prescribed in order to ensure an effective concentration in the affected area Vasodilators, such as Priscoline or Roniacol, may be of value. Salicylates combined with a barbiturate may be prescribed for relief of pain

Dry gangrene is ordinarily painless and spreads slowly, whereas moist gangrene is painful and spreads rapidly As pointed out by Allen and his co-workers (1955), diabetic lesions are likely to be moist and infected Furthermore, there is always the possibility that dry gangrene will be self-limiting, ending in spontaneous amputation of the dead part and arresting the progress of the gangrene It is advantageous, therefore, to keep dry gangrene dry and to attempt drying of moist gangrene

REFERENCES

Allen, E V , Barker, N W , and Hines, E A Peripheral Vascular Diseases, ed 2, Philadelphia, 1955, W B Saunders Co , p 231

Cady, J B , and Morgan, W S Treatment of Chronic Ulcers With Chlorophyll, Am J Surg 75 562-569, April, 1948

Kulowski, Jacob, and Perlman, Robert Skeletal Changes in Malum Perforans Pedis, Arch Surg 32 1-21, Jan , 1936

Lowrie, W L , Redfern, W E , and Brush, B E The Diabetic Foot, Clinical Orthopaedics, No 6, Philadelphia, 1955, J B Lippincott, Co , pp 173-181

Lucca, Domingo Management of Leg Ulcers, J Internat Coll Surg 25 718-726, June, 1956

Milberg, Irving Gelfoam in Stasis Ulcers, Scope 4 17-19, No 10, 1956

Smith, E M Familial Neurotrophic Osseous Atrophy, J A. M A 102 593-595, Feb 24, 1934

Tocantins, L M , and Reimann, H A Perforating Ulcers of Feet, With Osseous Atrophy, J A M A 112 2251-2255, June 3, 1939

Gangrene in the Diabetic Patient

The diabetic patient with gangrene of the foot (Fig 86) should rest Control of diabetes is in the province of the internist Conservative management should be exhausted in all cases in which gangrene is limited to a toe or toes When gangrene of the foot progresses rapidly and the local and systemic symptoms become alarming and do not respond to conservative measures, then amputation is imperative

Fig 86 —Diabetic gangrene

Lowrie and his associates (1955) suggested the following general criteria for differentiating between terminal and nonterminal diabetic gangrene

> if the skin of the foot is warm, elastic, of normal thickness, with some subcutaneous fat, and if pulsations can be felt in the dorsalis pedis and the posterior tibial arteries, it can be assumed that the body of the foot has a supply of blood, and healing could be expected without loss of the foot
>
> If the skin of the foot is thin, tightly applied to the tendons with absence of subcutaneous fatty tissue, colder than the other foot, and arterial pulsations are absent, it can be assumed that there has been gradual arterial occlusion above the ankle *

The foot should be left exposed to the air and protected by a foot cradle, the sides of the cradle should be padded to prevent further injury The position of the foot should be changed frequently to prevent the development of pressure sores Since the heel is the commonest area of pressure gangrene, a doughnut pad applied around the heel, held on by gauze around the foot and ankle, is a helpful measure

*From Lowrie, W L, Redfern, W E, and Brush, B E The Diabetic Foot, Clinical Orthopaedics, No 6, Philadelphia, 1955, J B Lippincott Co, pp 176-177

I Benign new growths
  A Nonosseous
    Lipoma
    Fibroma
    Giant cell tumor (benign)
    Xanthoma
    Angioma
    Melanoma
    Glomus tumor
  B Osseous
    Osteoma
    Chondroma, osteochondroma
    Bone cyst
II Malignant new growths
  A Carcinomas
    Epithelioma
    Melanocarcinoma
  B Sarcomas
    Osteogenic
      Osteosarcoma
      Chondrosarcoma
      Chondromyxosarcoma
      Ewing's sarcoma
    Soft tissue
      Fibrosarcoma
      Synovioma
      Subungual melanoma
      Kaposi's sarcoma
      Congenital sarcoma

BENIGN NONOSSEOUS TUMORS

Lipoma

The benign lipoma is composed of fat cells within a circumscribed lobulated mass, but it may arise where no fat exists Lipomas are common They attract little attention unless they become large or occur in an unusual site They appear beneath fascia and periosteum and in muscles and joints They form on subcutaneous tissues as soft, movable, either solitary or multiple masses The mass of fat cells may grow enormously even when the body fat is diminishing Lippsett and Johnson (1954) reported a case of multiple lipomas in a Negress, one 3 cm lipoma grew posterior to the left calcaneus and another was attached to the Achilles tendon Child (1955) reported a case of lipoma on the calcaneus of a man, aged 42 years, who experienced aching in the heel, aggravated by standing, running, or long-distance walking

Fibroma

Fibroma is a benign connective tissue growth It may form on any part of the foot It may be a true neoplasm or a reactive process resulting from friction or other trauma The traumatic type occurs over the posterior surface of the tendo achillis, just above its insertion A fibroma may form over the first metatarsal cuneiform joint, and occasionally it forms over a toe (Fig 87)

Giant Cell Tumor

Benign giant cell tumors *(osteoclastomas)* occur mostly in adolescents, at times at the proximal end of the fibula, rarely in the small bones of the foot The giant cell tumor used to be regarded as malignant Many types of tumors

CHAPTER SEVEN

Tumors*; Cysts; Exostoses

TUMORS AND CYSTS

THE FOOT AND ANKLE ARE COMMON SITES OF TUMOR FORMATION BECAUSE OF THE irritation from pressure induced by their weight-transmitting function and the frequency of trauma to the lower extremities Although the traumatic factor remains theoretical in the etiology of cancer, it is nevertheless likely that repeated trauma, when the predisposition already exists, may stimulate a benign growth, or stimulate one that is potentially malignant, to malignancy Malignancy even in the remote area of a toenail may produce a fatal malignancy anywhere in the body, especially in the lung Unless diagnosis is made early and treatment is instituted at once, amputation of the limb may be necessary as a lifesaving measure Every abnormal mass on the foot should be carefully studied grossly before removal and microscopically after removal

CLASSIFICATION OF TUMORS

No standard nomenclature of tumors of the foot has been adopted, although many classifications have been suggested Some observers classify new growths according to the site at which they occur, that is, their location in the body and the tissue in which they develop Some classify tumors histologically, others, according to whether tumors are benign or malignant Tumors of the foot are ordinarily benign (Coley and Higinbotham, 1939, Coley, 1953) Each pathologist has his preferred method of classification (Kulchar, 1944) For the purposes of this book, tumors of the lower extremities are discussed according to the following groupings

*Tumors and cysts are well covered in books on pathology, however, they are usually considered as a whole, without concentrated comprehensive pertinence to the foot

Aspiration of the tumor's thick orange-brown fluid yields large quantities of cholesterol, a characteristic that Minear considers pathognomonic Stout (1944) noted that the yellowish-brown color may shine through the epidermis, causing the lesion to be mistaken for a blue nevus or a melanoma

Angioma

An angioma (hemangioma), arising from lymph and blood vessels, only rarely occurs in the foot. The cause of this growth is not known, but apparently many hemangiomas are congenital Johnson and his associates (1956) reported that of their ninety-three cases occurring in the extremities, 33 per cent were said to be present at birth, 19 per cent were ascribed to trauma, and the remainder, to unknown causes The cavity of the growth may be filled with blood of currant jelly appearance Microscopically, the capillary type shows newly formed capillaries within sparce fibrous stroma, whereas the cavernous type shows large endothelium-lined spaces filled with blood

Angiomas are radiosensitive and benign Oughterson and Tennant (1939) believed that angiomas are of concern because of the local symptoms that they produce rather than because of the danger of malignancy. They recur after removal only if removal has been incomplete Usually angiomas are not diagnosed until after removal Kleinberg (1942) reported a case of benign capillary hemangioma in a young woman, aged 19 years, which involved the cuboid and external cuneiform bones and soft tissues of the sole

Melanoma

The benign type of melanoma, or *nevus*, has been observed on many white persons Usually little attention is paid to melanomas, particularly when the sites are inconspicuous as they are on the foot

The nevus may or may not be elevated in the skin, it varies from dark brown to black, is often covered with hair, and is usually small, although sometimes it may spread to great size. So-called juvenile melanomas are ordinarily benign Among 1,220 verified cases of malignant melanomas of childhood, Pack and Adair (1939) did not observe any instances of metastases

Melanomas on the feet should be watched because of pressure and trauma More than 17 per cent of Pack and his associates' (1952) series of pigmented moles were in the lower extremities Notwithstanding the slight possibility of their becoming malignant, there is always a chance that such a tumor is malignant at the outset Removal is therefore advisable Despite the uneventful course of most *nevi*, any sudden increase in size should suggest malignancy (Fig 88) The mass should be widely excised and the regional lymphatics dissected

Glomus Tumor

In the skin of the extremities, especially of the digits, are normal areas where the blood passes directly from artery to vein without passing through capillaries The channel through which the blood passes is surrounded by large *epithelioid*

contain giant cells, and the true giant cell tumor must be differentiated from bone sarcoma, malignant myeloma, and metastatic carcinoma, as well as from benign lesions, such as an osseous cyst and a nonosteogenic fibroma It is distinguished from nonosteogenic fibroma, chondroblastoma, and bone cyst by the presence of giant cells and stroma cells in the tumor Stroma cells are characteristic of the neoplasm

Amputation is indicated when at the onset of the growth there is certain evidence of breaking through the cortex and of invasion of the soft tissues and adjacent bone structure In a series of cases of giant cell tumors, Jaffe and his associates (1940) observed that about half responded favorably to treatment, about a third recurred after surgical excision or irradiation, some required amputation, 15 per cent proved to be malignant and tended to metastasize

Fig 87 —Fibroma of distal end of fourth left toe

Xanthoma

Xanthoma is sometimes referred to as *giant cell tumor of the tendon sheaths*, or as a *variant giant cell tumor* Microscopically, it contains small and large giant cells and abundant hemosiderin The tumor is encapsulated and fibroid It is observed in tendon sheaths about the fingers, ankles, and toes In some instances, pressure from the overlying tumor may produce localized atrophy of the bone, or the tumor may even invade the bone

Minear (1951) reviewed reports of seventy-five cases of xanthoma of the joint and described five more cases in which two of the tumors were located in the knee, two in the ankle, and one in the fibula In the case of ankle tumor, erosion extended to the tibia and talus and required amputation through the upper third of the tibia

Aspiration of the tumor's thick orange-brown fluid yields large quantities of cholesterol, a characteristic that Minear considers pathognomonic Stout (1944) noted that the yellowish-brown color may shine through the epidermis, causing the lesion to be mistaken for a blue nevus or a melanoma

Angioma

An angioma (hemangioma), arising from lymph and blood vessels, only rarely occurs in the foot The cause of this growth is not known, but apparently many hemangiomas are congenital Johnson and his associates (1956) reported that of their ninety-three cases occurring in the extremities, 33 per cent were said to be present at birth, 19 per cent were ascribed to trauma, and the remainder, to unknown causes The cavity of the growth may be filled with blood of currant jelly appearance. Microscopically, the capillary type shows newly formed capillaries within sparce fibrous stroma, whereas the cavernous type shows large endothelium-lined spaces filled with blood

Angiomas are radiosensitive and benign Oughterson and Tennant (1939) believed that angiomas are of concern because of the local symptoms that they produce rather than because of the danger of malignancy They recur after removal only if removal has been incomplete Usually angiomas are not diagnosed until after removal Kleinberg (1942) reported a case of benign capillary hemangioma in a young woman, aged 19 years, which involved the cuboid and external cuneiform bones and soft tissues of the sole

Melanoma

The benign type of melanoma, or *nevus*, has been observed on many white persons Usually little attention is paid to melanomas, particularly when the sites are inconspicuous as they are on the foot

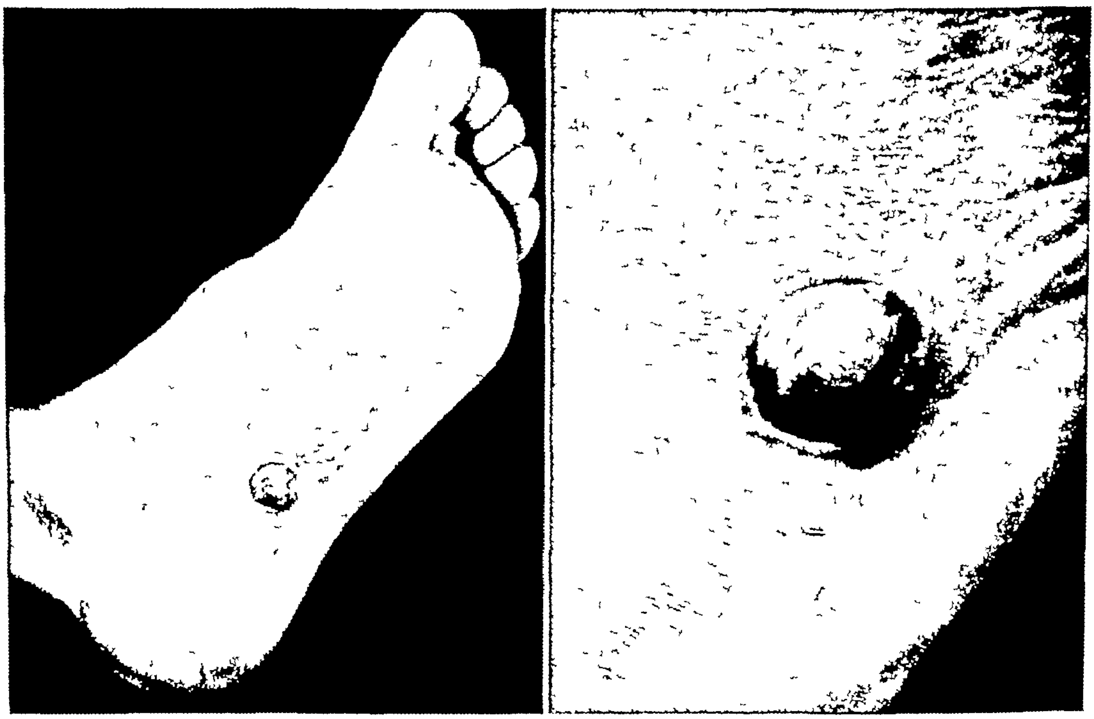
The nevus may or may not be elevated in the skin, it varies from dark brown to black, is often covered with hair, and is usually small, although sometimes it may spread to great size. So-called juvenile melanomas are ordinarily benign Among 1,220 verified cases of malignant melanomas of childhood, Pack and Adair (1939) did not observe any instances of metastases

Melanomas on the feet should be watched because of pressure and trauma More than 17 per cent of Pack and his associates' (1952) series of pigmented moles were in the lower extremities Notwithstanding the slight possibility of their becoming malignant, there is always a chance that such a tumor is malignant at the outset Removal is therefore advisable Despite the uneventful course of most *nevi*, any sudden increase in size should suggest malignancy (Fig 88) The mass should be widely excised and the regional lymphatics dissected

Glomus Tumor

In the skin of the extremities, especially of the digits, are normal areas where the blood passes directly from artery to vein without passing through capillaries The channel through which the blood passes is surrounded by large *epithelioid*

or *glomus cells* Small nodular masses sometimes form in those areas and are exceedingly painful or of burning sensation The masses, which King (1954) regards as malformations rather than as neoplasms, consist of a network of blood vessels within a capsule of typical glomus cells The tumors occur mostly in the skin and subcutaneous tissues, but they may originate in muscles, tendons, bone, and even in deeper parts of the body Abramson (1956) observed them in the nailbeds of fingers and toes and in the palm of the hand and the sole of the foot When located in the fingers and toes, they do not invade other structures, and they remain benign They recur only when removal has been incomplete Ottley (1942) observed that the tumor was solitary in all but thirteen instances of 173 cases



A B

Fig 88 —*A*, Malignant melanoma on plantar surface of foot *B*, Enlargement, same case as shown in *A* (From Wigley, J E M Med Illus 8 607, Sept, 1954)

I saw a patient who had violent pain on the lateral of a lesser toe, for which there was no rational explanation nor was there response to palliative therapy On sectioning of the excised subdermal tissue in the painful area, a glomus pattern was disclosed, excision brought complete relief In two other cases in which a diagnosis of neurofibroma *(neurilemmoma)* of the medial plantar nerve had been made, symptoms resembled those of an exaggerated Morton's neuralgia Histologic study showed glomus tumor The lesions are benign Complete extirpation brings immediate relief

The diagnosis is based on severe pain or agonizing throbbing in a small area of the foot where pathologic disturbance is not clinically demonstrable

Even pressure of bedding can produce excruciating pain which may not be localized but may be of a neuralgic radiating type The growth may exist for years before it is disclosed by earnest evaluation of symptoms The definitive diagnosis may not be made until the removed specimen has been examined Ottley (1942) reported two cases of needless amputation as a result of faulty diagnosis

BENIGN TUMORS COMPOSED OF CARTILAGE OR BONE

The term *osteogenic*, as Warnick and Thomson (1954) pointed out, has two meanings· *bone-forming*, as the term is applied by histologists, and *arising from bone*, as Ewing employed it The former meaning is intended here As Stout

Fig 89

Fig 90

Figs 89 and 90 —Osteoma of second metatarsal

(1944) emphasized, "extraskeletal formation of bone can take place about any group of mesenchymal cells" Furthermore, this formation can occur in granulation and scar tissue and in calcified areas of necrosis or degeneration when they are invaded by granulation tissue But those are not neoplastic processes

Osteoma

Osteoid osteoma is a benign tumor which often occurs in the foot It is of two types (1) a type secondary to trauma, such as subungual exostosis, calcaneal spur, and exostosis on the dorsum of the first metatarsal, first cuneiform, and

Fig 91

Fig 92

Fig 91 —Osteoma of second and third metatarsals

Fig 92 —Osteoma of first metatarsal

navicular bones, and (2) an uncommon idiopathic type which may assume bizarre patterns (Figs 89 to 92)

Jaffe and Lichtenstein were the first to designate the tumor *osteoid osteoma* in 1942 They discussed sixty-two cases Osteoid osteoma has become generally accepted as an entity, but, according to Moberg (1951), whether it should be classified as a tumor depends on the definition of the term Usually it is described as a benign neoplasm. Although most observers agree that *osteoid osteomas* are benign, not all agree that they are neoplasms.

Fig 93 —*A*, Osteochondroma of second proximal phalanx *B*, After removal of growth

Chondroma; Osteochondroma, Chondromyxosarcoma

Chondromas are said to be the commonest of benign tumors arising in or near the bones of the foot They may be either cartilaginous or intermixed with bone These tumors may involve the bone structure or the bone itself (Figs 93 to 95) or lie adjacent to the bone (Figs 96 to 98) They often arise in the small bones of the hands and feet and sometimes grow to unsightly crippling masses Copeland (1956) suggested that cartilaginous tumors may arise from an island of cartilage persisting within the medullary substance, in subperiosteal tissue in the area of attachment of tendons to tuberosities, and in periarticular

Fig 94

Fig 95

Fig 94 —Osteochondroma of neck of talus

Fig 95 —Osteochondroma of third metatarsal and third toe (Courtesy Dr Ronald Tanner)

A *B*

Fig 96—*A*, First roentgenograms taken Suggested possible sarcoma of first proximal phalanx Left, Dorsiplantar view Right, Oblique view *B*, Medial view taken later shows tumor adjacent to bone Diagnosis osteochondroma adjacent to first proximal phalanx

Fig 97—Osteochondroma under middle three metatarsal heads

areas. At any of those sites, as well as within joint synovium, skeletal embryonal tissue can persist and undergo tumor formation

Osteochondroma is a common benign lesion arising at the site of insertion of certain tendons, usually near the ends of the long bones, but sometimes in the small bones of the feet.

Geschickter and Copeland (1949) included among the precartilaginous connective tissue tumors or chondromas, *osteochondromas,* and *chondromyxosarcomas* —all three types representing different stages of the same pathologic process The most primitive, the chondromyxosarcoma, is malignant, the chondroma is potentially malignant when recurrent, and the osteochondroma is essentially benign. After he studied a large series of such tumors, Copeland (1956) observed that 5 per cent showed malignant changes and advised immediate excision, perhaps amputation, when a long quiescent chondroma suddenly begins to grow.

Fig 98 Fig 99

Fig 98 —Osteochondroma adjacent to fifth middle phalanx
Fig 99 —Chondromyxoma of first proximal phalanx (Courtesy Dr James Brightwell)

Both solitary and multiple *chondromyxomas* are frequent in the small bones of the feet (Fig 99), as well as in long bones They may be cured by curettage and chemical cauterization Although malignant growth is rare in the phalanges and metatarsal bones, Coley (1949) warned that insidious transformation of chondromas to chondrosarcomas is always a possibility

Bone Cysts

It is possible to confuse bone cysts with giant cell tumors. When giant cells appear in bone cysts, they do not indicate a giant cell tumor, for giant cells lying among stromal cells in a giant cell tumor lack intercellular substance The cyst has a scattering of giant cells in the fibrovascular tissue, honeycombed by vascular spaces, the giant cell tumor, on the other hand, has many multinucleated giant cells in the vascular spindle-celled stroma Moreover, as noted by Miles and Degenshein (1956), bone cysts tend to be symmetric, whereas the giant cell tumor enlarges asymmetrically The cortex usually remains intact in the bone cyst, but in the giant cell tumor the cortex is often involved and perforated.

Bone cysts may develop in any bone of the foot (Figs 100 to 102). If a bone cyst occurs on a weight-bearing area, pain and inflammatory changes may arise early, whereas when it occurs on a nonweight-bearing area, the condition may

Fig 100 —Solitary cyst of cuboid

be symptomless and go undiagnosed unless it is disclosed when roentgenograms are taken for other reasons Latent solitary cysts of the calcaneus (Figs. 103 and 104) have been reported Kingsbery (1957) reported eight cases He treated the patients surgically I have seen an equal number, however, in my cases only three of the cysts became symptomatic, requiring surgical extirpation The cortex about the cyst is sclerosed and forms a complete round or oval shell around the degenerated area The cystic area is always weakened, thereby subject to spontaneous fracture After spontaneous fracture, the cyst may become obliterated during the healing of the fracture In symptomatic cases, the cyst should be curetted and bone chips implanted

Bone cysts present few signs and symptoms, they may be disclosed in roentgenograms taken for other purposes Pathologic fracture may be the earliest symptom A small rounded area of bone destruction with well-defined margins may be observed to lack the trabeculations of giant cell tumors Usually the cyst contains a clear yellowish fluid

Fig 101

Fig 102

Fig 101 —Bone cyst on tuberosity of calcaneus

Fig 102 —Bone cyst on tuberosity of calcaneus with coincidental extensive osteoporosis of entire foot

The *aneurysmal bone cyst* is a benign, solitary, localized, and expanding fibrous lesion presenting a honeycombed appearance and having a large dilated, networklike vascular bed It occurs mostly in children and young adults The aneurysmal bone cyst may involve most bones of the body, including at times the tarsals and metatarsals (Figs 105 and 106) Although generally benign (Jaffe, 1950), the cysts can be destructive, requiring irradiation and extensive reconstruction Lichtenstein (1952, 1953) reported a case (1957) of aneurysmal cyst of the first metatarsal which, after three attempts at surgical extirpation, finally required amputation below the knee Roentgenograms in such cases show

Fig 103

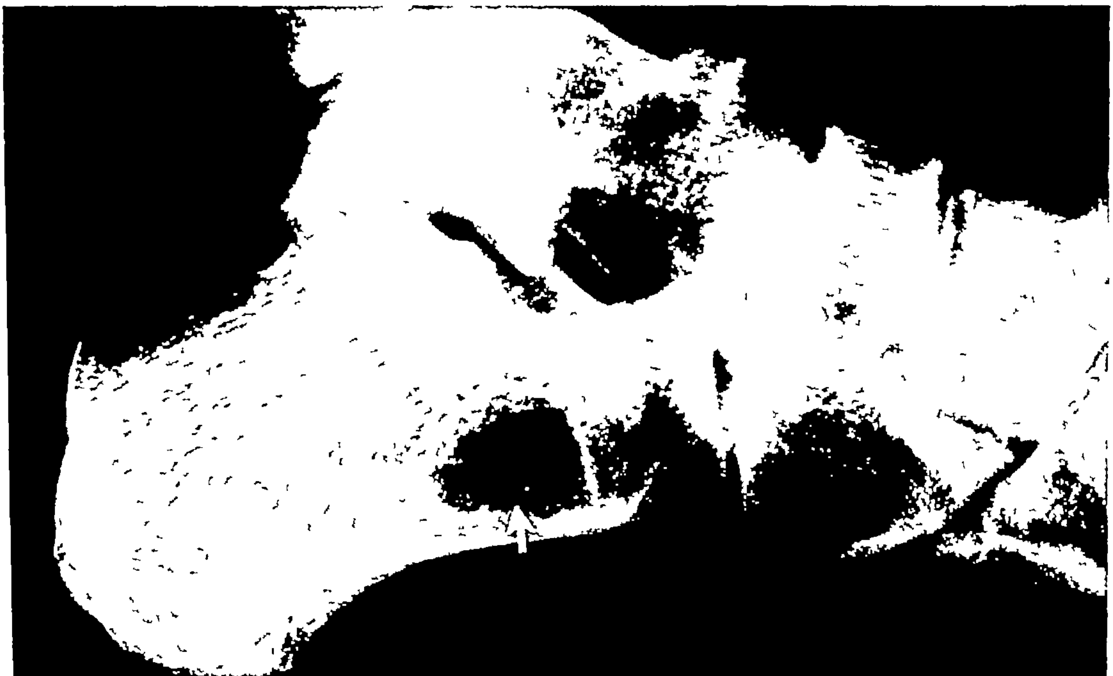


Fig 104

Fig 103 –Solitary cyst of head of calcaneus

Fig 104 –Solitary cyst of head of calcaneus, symptomatic

an eccentrically located cystic area which may expand the cortex. The cyst has a sharp rim of periosteal bone and a translucent center with trabeculations, coarse at the periphery and finer toward the center. The cyst contains stagnant unclotted blood and sometimes bony fibers, in addition to osteoid spicules. The treatment of choice is either complete excision, if practicable, or thorough curettage

Fig 105

Fig 106

Fig 105 —Aneurysmal bone cyst of first cuneiform

Fig 106 —Aneurysmal bone cyst of third metatarsal (From Sherman, R S , and Soong, K Y Aneurysmal Bone Cyst Its Roentgen Diagnosis, Radiology 68 61, Jan , 1957)

MALIGNANT TUMORS

The most exhaustive measures, including a biopsy, should be taken to rule out the possibility of malignancy whenever there is the slightest suggestion that the tumor is not benign Cater (1953) has made this careful histologic distinction between benign and malignant tumors Tissue arrangement in the benign tumor is similar to normal tissue, whereas in malignant growths the tissues differ greatly from normal tissue, even to complete loss of cell arrangement Although in the benign form there is a normal relationship of epithelial elements to connective tissue stroma, in the malignant tumor the supporting stroma is proliferated by malignant cells, the basement membrane is not intact, and the polarity of cells is often reversed. The benign tumor does not metastasize, the malignant tumor spreads by blood stream and lymphatics to produce local or distant metastases In the benign tumor, the blood supply increases moderately to prevent necrosis, in the malignant type, the blood supply is rich, but growth of these tumors outstrips that supply and produces necrosis

TABLE 1 DIFFERENTIAL DIAGNOSIS BETWEEN BENIGN AND MALIGNANT TUMORS

FEATURE	BENIGN	MALIGNANT
Capsule	Present	Absent
Recurrence	No	Yes
Blood supply	Poor	Rich
Relation to surrounding tissue	Not adherent	Adherent
Outline	Regular	Irregular
Metastasis	No	Yes
Cells	Adult	Embryonic
Growth	Slow	Rapid
Influence on life	Little or none	Nearly always fatal

Because the foot is essentially composed of connective tissue, most primary neoplasms of the foot (Coley, 1949) are connective tissue tumors The plantar skin is a highly specialized epithelium, there are few neoplasms in that area Pascher and Sims (1954) reported two cases of epithelioma of the sole

Carcinomas

Carcinomas rarely occur in the foot When they do, they are usually primary, either squamous or basal cell carcinomas Secondary carcinomas in the foot arising by metastasis from some other part of the body are hardly ever reported

Epithelioma –Epitheliomas or squamous cell carcinomas of the extremities comprise about 1 per cent of carcinomas of all parts of the body An extensive study was made by Browne (1953) of such reported cases as well as of a series of 511 cases at the Mayo Clinic from 1908 to 1946 Of the Mayo Clinic series, 164 (32 per cent) were in the lower extremities and 347 (68 per cent) in the upper, 55 cases were in the foot itself

Epitheliomas arising from the sites of warts and scars are frequently reported. Charache (1939) reported the distribution of carcinoma in thirty-two cases. In order of frequency, the sites were dorsum of hand and wrist, leg, arm, forearm, foot, fingers, and thighs, only rarely the toes. In four instances the growth originated in warts, and in three, in scars from burns received from twenty-four to thirty-five years earlier.

Schreiner and Wehr (1933) reported a squamous cell epithelioma originating at the undersurface of the fourth toe on the site of a corn, which involved both the fourth and fifth toes and the dorsum and sole of the foot.

Epithelium cuniculatum, a variety of squamous carcinoma peculiar to the foot, has been discussed by Aird and his associates (1954). They reported the case of a man, aged 64 years, who had had a bulbous mass on the sole, covered with skin containing many sinuses, most of which opened at the apex of a separate bulge. Aird suggests that a low-grade, highly keratinizing squamous carcinoma developed on the ball of the foot and that repeated pressure forced portions of the growth into the soft tissues, probably accelerating growth.

Melanocarcinoma.—Melanocarcinomas arising from nevi of the soles and palms are frequently observed. Allen and Spitz (1953) described the clinical and histologic features of this growth in a series of 934 cases occurring on the palms, soles, and genitalia. Two-thirds of the patients were 31 to 60 years of age. Juvenile melanoma is often confused with this malignant growth. Unless landmarks have been destroyed, differentiation between the primary and metastatic tumor is not difficult. Allen and Spitz thought that the junctional nevus rather than the intradermal nevus was the source of melanocarcinomas of the skin, except those arising from the blue nevus, however, only a small percentage of junctional nevi become malignant. The authors stated that because of the vulnerability of moles in the soles and palms, moles at these sites should be excised whenever feasible, preferably before puberty. Not infrequently, however, lesions in adults are diagnosed as melanocarcinoma, whereas in reality they are benign juvenile melanomas persisting into adulthood. Approximately one of every ten melanocarcinomas apparently are superimposed on compound nevi or juvenile melanomas.

Another series of twenty-five cases of melanocarcinoma of the plantar surface of the foot was reported by Decker and Chamness (1951). Nine of their patients reported a mole present from birth, in the remainder, one mole had been present from three to twenty years. Six patients reported plantar injuries, three from nail punctures. Local therapy had been received before hospital admission in seventeen cases. In no instance was a correct diagnosis made, malignancy was suggested in only five cases. Some of the melanocarcinomas were shaded from brown to black, others were only slightly colored, and in six instances melanin granules were not observed.

A biopsy is required for all nevi on the sole of the foot whether pigmented or not. If the plantar lesion proves microscopically to be a melanocarcinoma, radical operation is necessary. Local excision is adequate when the lesion is less than 2 cm in diameter and does not show a tendency to deep fixation.

Subungual Melanoma.—Pack and Adair (1939) reviewed sixty-nine reported cases of subungual melanoma and described sixteen others Of the total, thirty-four were located in the great toes, three in other toes

The growth is usually in the form of a black fungating ulcer, involving the nail sulcus and matrix and elevating the nail A black border along the edge of the nail is pathognomonic Subungual melanomas contrast with benign forms which do not break through the nail Prognosis depends on whether there is metastasis The percentage of cures is higher than for malignant melanomas elsewhere Prompt amputation should follow diagnosis Pack (1939) advised amputation first and dissection of the lymph nodes one or two weeks later if there is gross evidence of metastatic involvement, and amputation six weeks after excision if dissection was prophylactic

Sarcomas

Sarcomas are composed of cells mainly derived from connective tissue Microscopic study shows giant cells, spindle cells, and differentiated cells, according to the type of sarcoma Most sarcomas occur in early life They are among the most malignant neoplasms known Usually they are rapidly destructive of bone and metastasize to the lungs An early diagnosis is essential, because even a short delay may mean the difference between life and death

Osteogenic Sarcoma (Osteosarcoma).—Osteosarcoma is the commonest type of sarcoma It is a specialized connective tissue tumor containing osseous elements and arises in the interior of bone Jaffe (1956) stated that a malignant bone sarcoma without evident osteogenesis is a fibrosarcoma, not an osteosarcoma, however, osteoid and osseous tissue may be present in either small or great amounts The cell type is osteoblastic and readily recognized by its intercellular substance About three-fourths of the tumors occur between the ages of 10 and 30 years and mostly in boys and men Ackerman and del Regato (1954) found that 70 per cent occur in the lower extremity, usually at the end of the shaft of the long bones Since about 75 per cent of osteosarcomas metastasize to the lung, roentgenologic study should be made in each case before treatment of the primary neoplasm

Schreiner and Wehr (1933) described osteogenetic sarcoma in the foot of a young girl, aged 19 years Following irradiation, the lesion remained healed for four years, then the scar, irritated by the shoe, showed a bluish tinge Amputation of the leg was finally necessary The patient died eight and one-half years later from metastases to the lungs and viscera Another case occurred in the os calcis of a youth, aged 17 years He died two years later of extensive metastases

Meyerding (1944) reported a case of multiple metatarsal fractures of the right foot associated with malignant osteogenic sarcoma in a youth, aged 15 years

Chondrosarcoma—The chondrosarcomas comprise the largest group of tumors among osteogenic sarcomas They contain cartilage in association with a type of myxomatous connective tissue, indicating an origin analogous to the

benign exostoses and chondromas Some chondrosarcomas grow slowly, some seem to arise spontaneously, often they grow to a large size

Chondrosarcoma of the calcaneum is rare Geschickter and Copeland (1949) reported three among eighty-three cases of chondromyxosarcoma Durbin and Smith (1955) reported chondrosarcoma of the calcaneum in a man, 61 years of age, who had had a painful heel as a result of an injury thirty-two years before Longstreth and has associates (1948) reviewed ten reported cases in the talus and added a case of their own observed in a man, aged 62 years

Ewing's Sarcoma.—Ewing differentiated the tumor that bears his name from other sarcomas and classified it as an *endothelial myeloma* It is highly malignant, almost always fatal, and occurs in about 15 per cent of malignant tumors of bone The growth commonly involves a lower extremity, Geschickter and Copeland (1949), in an analysis of 122 cases, found sixty-six were in this extremity Additional cases have been reported by Castleman (1955), in the calcaneum of a girl, aged 12 years, by Roaf (1952), in the cuneiform bone of a boy, aged 14 years, and by Cohen and his associates (1953), in the talus of a boy, aged 3 years

The clinical history is characteristic, nevertheless, Ewing's sarcoma is frequently mistaken for osteomyelitis The patient is usually of an age under 16 years and only rarely over 30 In many instances there is a history of trauma which was followed shortly by pain, swelling, and local heat The roentgenogram shows diffuse involvement of the bone and a combination of bone formation and bone destruction

The tumor has shown prompt response to roentgen therapy in numerous instances, the growth may disappear for a time but then recur Surgical excision is the only alternative treatment

Fibrosarcoma.—Fibrosarcoma is a highly malignant tumor which occurs at any age, but mostly between the decades of 30 and 60 Because changes in the bone are not demonstrable roentgenographically, in most instances by the time the tumor is recognized, the fibrosarcoma has already metastasized, as illustrated by the following personal case A man, aged 42 years, reported a rapid increase, within six months, in the size of his left foot No other symptoms, not even pain, interfered with the use of the foot Two physicians whom he had consulted did not consider the enlargement serious and advised watchful waiting When I first examined the affected forefoot, it was half again as large as the other foot, it was nodular but had no freely movable masses Biopsy revealed a fibrosarcoma Amputation of the leg at the hip was performed The patient was last seen by me five years postoperatively

Synovioma—Willis (1952) described two types of synovioma (1) a benign fibroblastic giant cell growth, which in most instances arises from tendon sheaths and often is called *fibroma, giant cell tumor*, or *xanthoma*, already discussed, and (2) rare malignant growths predominantly of cystic papillary structure with synovia-filled spaces, arising from the capsule of large joints and bursae He observed that the benign form usually is solitary and appears in young and middle-aged adults, especially in the hands rather than in the feet Personal experience

and confirmatory consultations with pathologists* dictate a contrary conclusion, namely, *synoviomas are always malignant,* notwithstanding published reports of benign types King (1941) emphasized that most malignant synoviomas arise in the synovia, others have classified synoviomas according to the predominant cell found within them, for instance, Willis stated that malignant tumors of synovial origin may be solid anaplastic spindle cell or pleomorphic cell sarcomas in which synovial differentiation is indefinite or lacking, or they may be cystic, or papillary growths of pseudoepithelial structure, often containing gelatinous synovial secretion The anaplastic type is commoner than the cystic or papillary growths Lichtenstein (1955), who regarded the cytologic hallmark of synovium as the tendency of its primitive or mesenchymal spindle connective tissue cells to line spaces and ramifying clefts, believed that they are widely invasive, particularly in the sole of the foot

Synoviomas occur in young adults, mostly in the extremities, particularly the lower ones, about half of those in the lower extremities occur in the area of the knee Because of their smallness and apparent encapsulation, usually without roentgenologic or microscopic evidence, they are not regarded as serious and may not be treated for several years If untreated, or inadequately treated and there is recurrence, the tumor may become aggressive, extending to adjacent bones and metastasizing to the lungs The tumor may recur after five or ten years or longer Lichenstein (1955) says that a ten-year survival does not indicate permanent cure Surgical removal alone is not curative, adequate excision and postoperative irradiation may be lifesaving, but amputation of the limb is made necessary by a rapidly growing, invasive malignant synovium Even then, prognosis must be guarded Allen and his co-workers (1955) described one case in the arch of the foot and six in the sole All patients had had amputations, but metastasis ensued Six of the seven were known to have died Pack and Ariel (1950) reported sixty cases of malignant synovioma

Kaposi's Sarcoma.—Kaposi first described in 1872 the rare soft growth that bears his name It is characterized by a cutaneous tumor which usually begins on the distal portion of the lower extremity The tumor appears initially as a small brown, blue, or red macule Progression is manifested by the formation of other macules, papules, nodules, and plaques (Fig 107) The nodules vary in size and in number Oughterson and Tennant (1939) stated that its histologic pathology is so certain that examination of the lesion at any of its various stages of development establishes the diagnosis

As a number of lesions coalesce and become extensive, the limb gives the appearance of elephantiasis Itching is the commonest symptom, pain may be severe Untreated, the lesion may lead to death in five to ten years

Congenital Sarcoma.—Congenital sarcoma of the extremities is rarely reported Fahey and Bollinger (1953) discussed a case in the foot of a 3-month-

*Ernest Nora, Sr, M D, Chief Pathologist, Columbus Memorial Hospital, Chicago, Edwin F Hirsch M D Chief Pathologist, St Luke's Hospital, Chicago, and Paul B Szanto, M D, Director of Pathology, Cook County Hospital, Chicago

old infant The mass was observed two days after birth It was excised but recurrence took place in four months Amputation was performed in the upper tibia, the child died seven months later Chamberlain and Lawrence (1941) reported a case, proved by biopsy, of spindle cell sarcoma in a 5-week-old baby, who at birth had had a growth the size of a golf ball on the sole of the left foot

Prognosis of Malignancies of the Extremities

Stout (1953) believes that the cure of malignant tumors is in the hands of the first therapist If his treatment fails, later attempts, however radical, are unlikely to be successful Pathologic interpretation of biopsy specimens and extensive consultation should always precede therapy

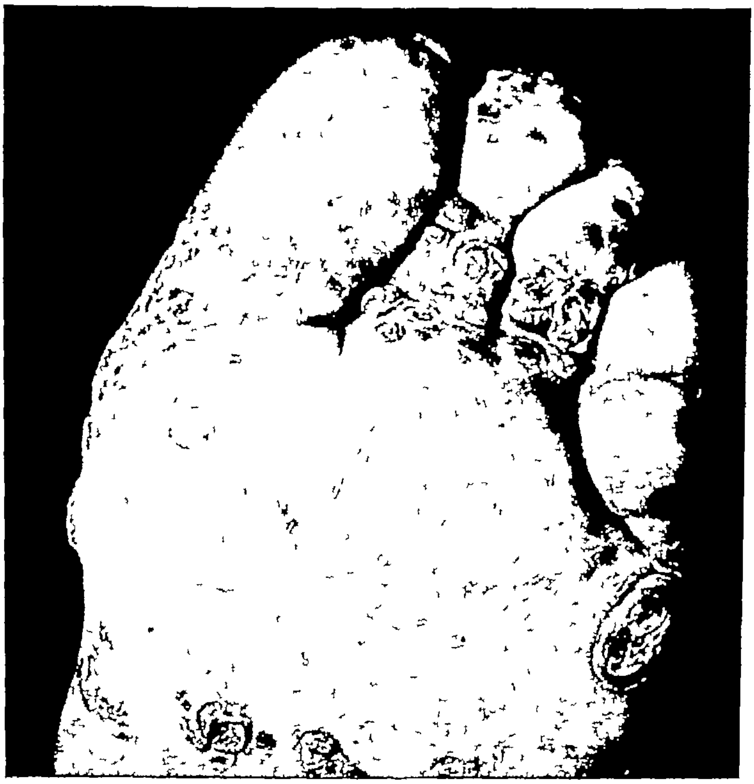


Fig 107 —Kaposi's sarcoma of foot (Courtesy Dr Marcus Caro)

EXOSTOSES

The foot is subject to exostosis more than any other skeletal part of the body, especially in the following areas (1) dorsum of the distal phalanges (subungual exostosis), (2) calcaneal spur, and (3) over the dorsum of the tarsals or metatarsals (Figs 108 and 109) The great toe is affected the most, whereas lesser toe involvement is in relation to their number, that is, the second toe more than the third, and so on Calcaneal spur is an exostosis at the tuberosity of the calcaneus where the fascia arises

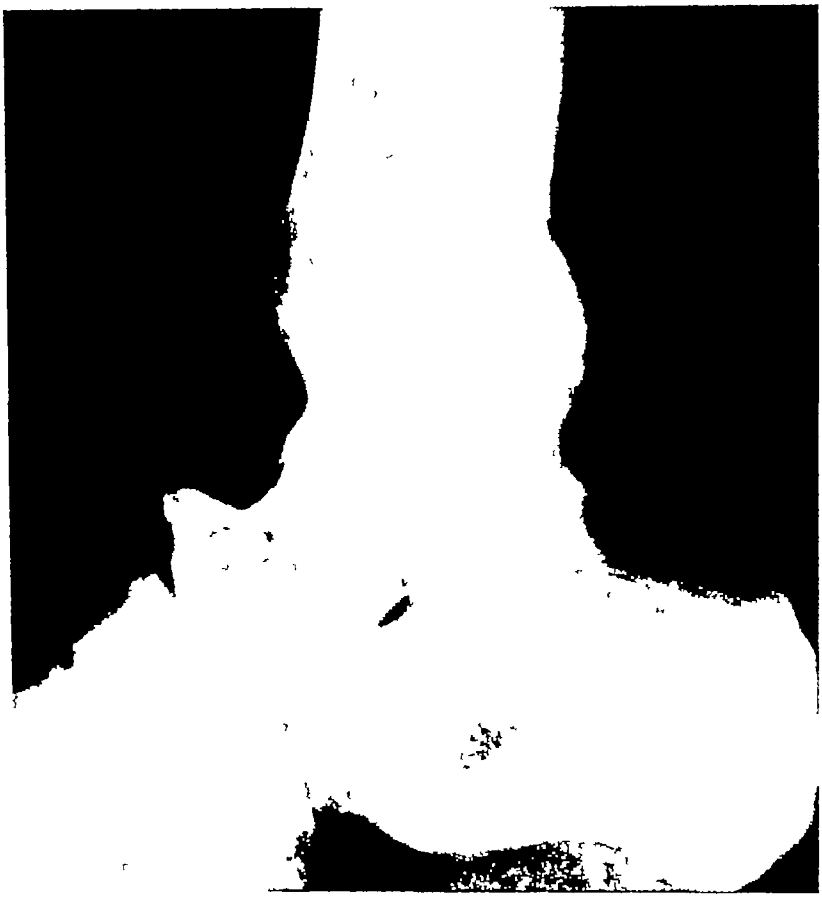


Fig 108 —Exostosis on dorsum of head of talus

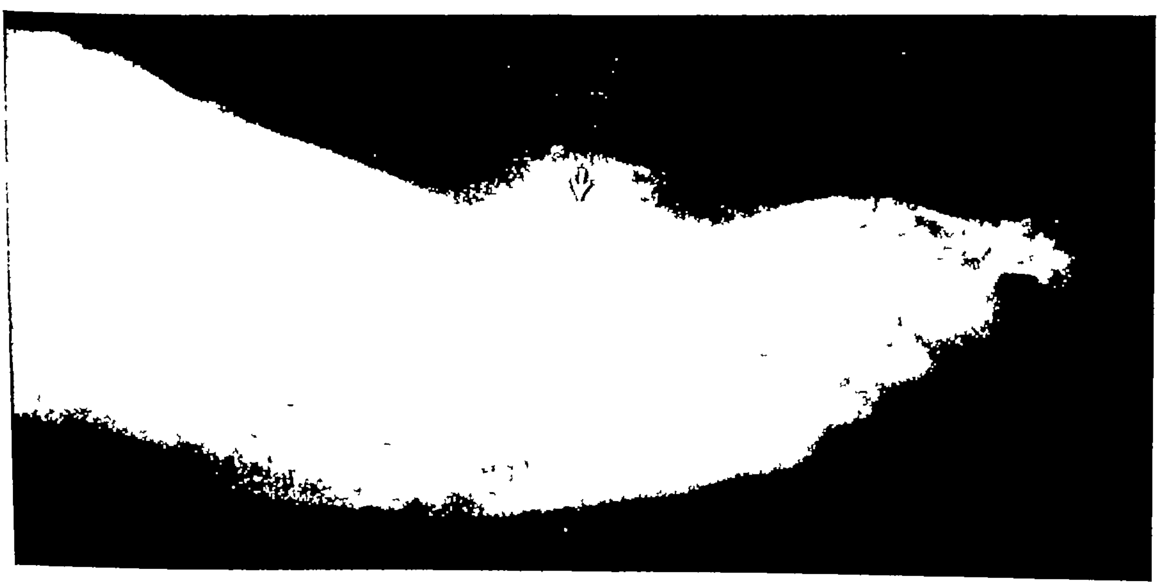


Fig 109 —Exostosis on dorsum of first metatarsal (Courtesy Dr Milo Turnbo)

Subungual Exostosis of Great Toe

Subungual exostosis of the great toe is an osteogenic growth over the dorsal surface of the distal phalanx of the great toe and is of two types: (1) those in which the dorsum of the distal phalanx is congenitally dome-shaped, representing a relative exostosis (Figs 110 and 111), (2) true exostoses occurring in bizarre shapes, often mushroomlike (Figs. 112 and 113) When the dome-shaped type is pronounced, it is subject to irritation and pressure from the boxing of the shoe, which causes further hypertrophy of the dorsum of the distal phalanx Secondary tinea infection of the nail and nail bed often ensues, which increases hypertrophy of the nail and the bed, thus making the deformity worse The highest point of the deformity is over the distal part and midline of the phalanx and is usually symmetric The deformity of the nail and the spongy hypertrophy

Fig 110

Fig 111

Fig 110 –Subungual exostosis over dorsum of distal tip of first distal phalanx

Fig 111 –Subungual exostosis on dorusm of neck of first distal phalanx

of the nail bed are painful. Sometimes when the diseased nail bed becomes secondarily infected, a chronic draining sinus forms under the anterior margin of the nail plate

True exostosis nearly always occurs on the tibial side of the distal portion of the distal phalanx and projects dorsally above the nail (Figs 114 and 115),

Fig 112

Fig 113

Fig 112 —Bizarre type of subungual exostosis

Fig 113 —Pole-shaped subungual exostosis.

so that the growing nail collides with the exostosis The surface of the exostosis is covered with a red glistening layer of granulation tissue which is adherent to the growth Exostoses occur at all ages, but mostly in children and adolescents, and equally in the two sexes The nail plate and the nail bed are normal except that they are absent over the exostosis

Etiology, Symptom of Pain, and Diagnosis.—The etiology is obscure Friction and pressure alone do not explain the presence of exostoses because they

Fig 114

Fig 115

Fig 114 —Massive subungual exostosis on tibial side of distal phalanx
Fig 115 —Small subungual exostosis on tibial side of distal phalanx

occur in young children (Figs 116 to 118) and overwhelmingly involve only the great toe

Painfulness is due to the collision of the distal growth of the nail with the exostosis which is in its path

Roentgenograms readily reveal the abnormality

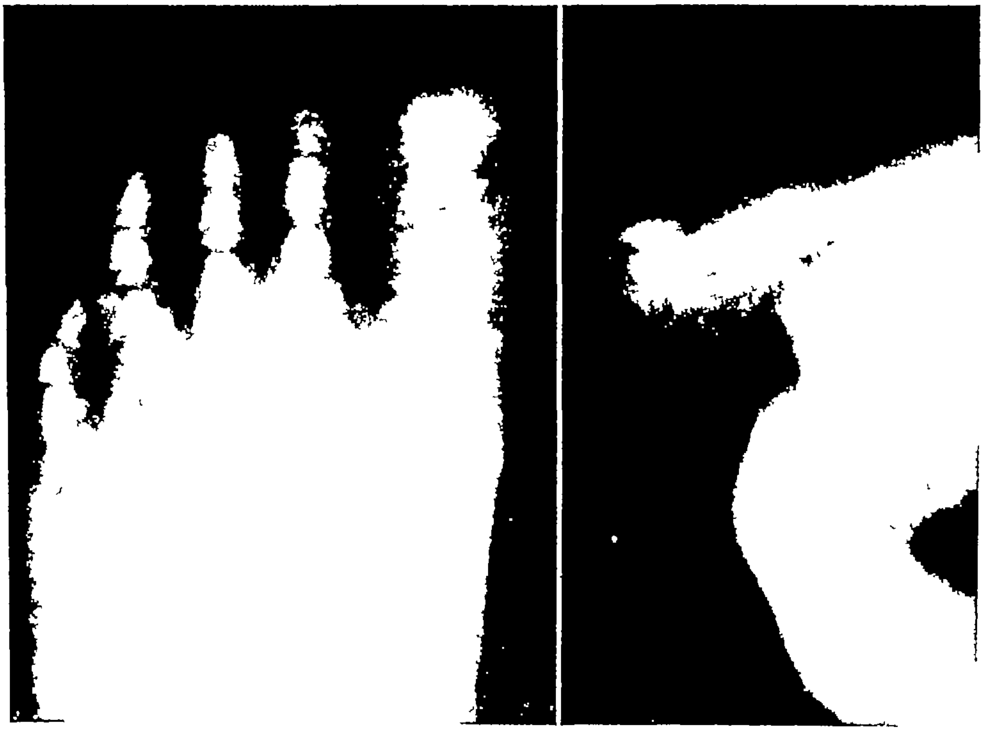


Fig 116 —Subungual exostosis in a 5-year-old child

Fig 117 —Subungual exostosis in an 8-year-old child

Recommended Treatment.—For the first type of exostosis in which hypertrophy includes the nail, nail bed, and the dorsum of the phalanx, the following procedure is satisfactory

1 Avulse the entire nail

2 Make a horseshoe incision around the margin of the nail bed

3 Denude the nail bed from the dorsum of the phalanx, proximally to the matrix This forms a flap which is elevated

4 Trim the undersurface of the flap of all hypertrophied tissue

5 Excise the dorsum of the distal phalanx with bone forceps, and flatten the surface with a rasp

Fig 118 —Subungual exostosis in an adolescent

6 Replace the flap of the nail bed to cover the surface of raw bone In some cases the flap needs to be sutured to the distal end or to the sides of the skin fold

7 Apply a compression bandage to prevent bleeding under the flap

Dress the toe at intervals of five to seven days until the wound becomes a dry scale In most cases the flap heals by first intention In the remainder, the flap sloughs, however, an epithelial cover forms in its path, and in six to ten weeks a rudimentary nail forms which rarely causes discomfort

For the second, or true, type of exostosis, the following procedure is the least traumatic and produces excellent results

1 Make a plantarwise incision, completely encircling the growth and freeing all surrounding soft tissues (Fig 119, *A*)

2 Amputate the growth at its neck with bone forceps (Fig. 119, *B*).

3. Smooth the area of amputation on the phalanx with a rasp.

4 Invaginate the skin fold and hold under the nail plate by a mattress suture (Fig 119, *C* and *D*).

The wound always heals by first intention As the nail grows, the toe soon becomes completely normal

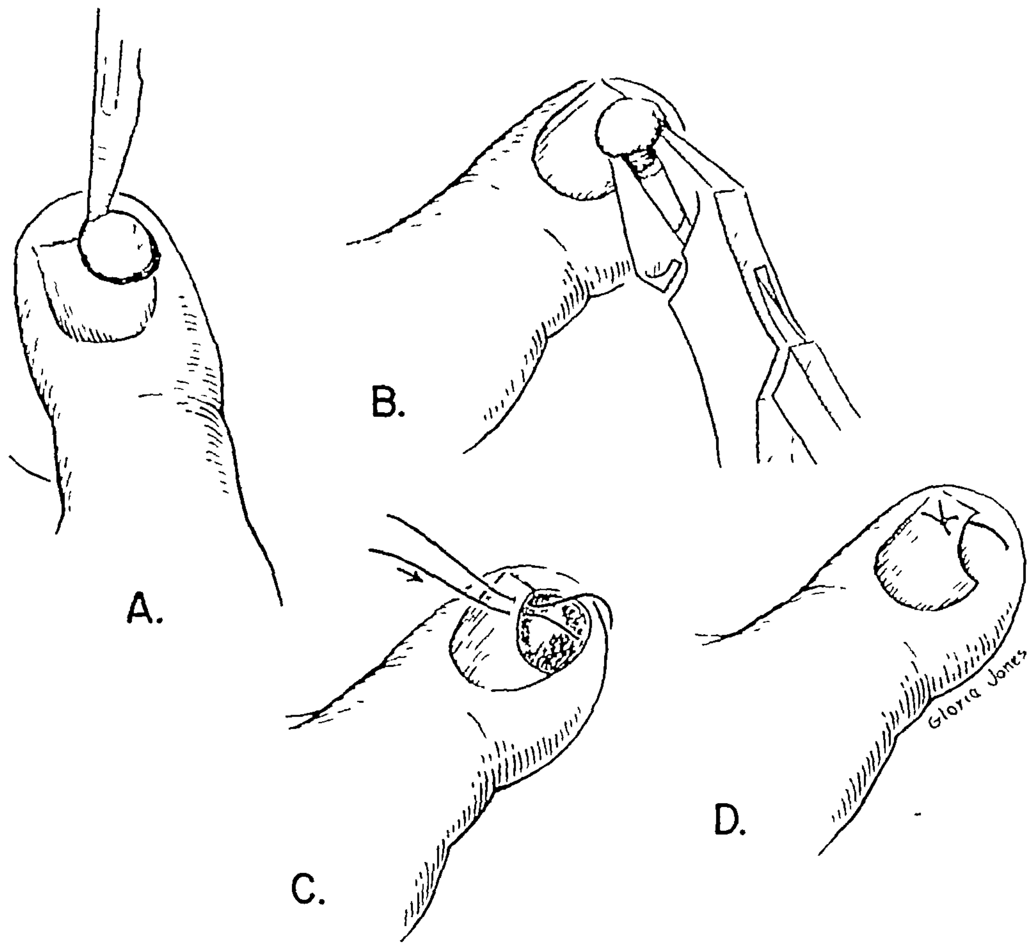


Fig 119 —DuVries' technique *A*, Incision completely encircling exostosis, *B*, Exostosis amputated *C* and *D*, Skin invaginated under nail by means of modified mattress suture through nail

Subungual Exostosis of Lesser Toes

Subungual exostosis of the lesser toes is common but rarely of the second type described (Fig 120) It is a cone-shaped deformity, largely due to hypertrophy of the nail bed as a result of tinea infection Both margins of the nail are inverted A bulbous mass forms under the distal portion of the nail The second toe is affected most The following procedure is recommended

1 Make a plantarwise incision around the nail plate inside the two lateral borders and the anterior lip. Let the incision penetrate to the plantar fat pad of the toe

2 Denude the nail plate from the nail bed to the matrix and elevate it

3 Retract the flap around the distal phalanx with Allis forceps to expose the hypertrophied dorsum of the distal phalanx

4 With sharp-pointed bone forceps, remove the entire dorsal surface of the distal phalanx

5 Invaginate the anterior portion of the skin flap and hold it under the anterior edge of the nail with a mattress suture as follows Bring the needle through the anterior edge of the nail downward, then through the anterior lip from the inside outward Return the suture, again through the nail plate close to the initial puncture and through the underside of the nail plate upward Draw the suture up snugly This will pull the anterior skin flap in under the anterior edge of the nail Similar sutures may be required at times to approximate the lateral edges of the incision

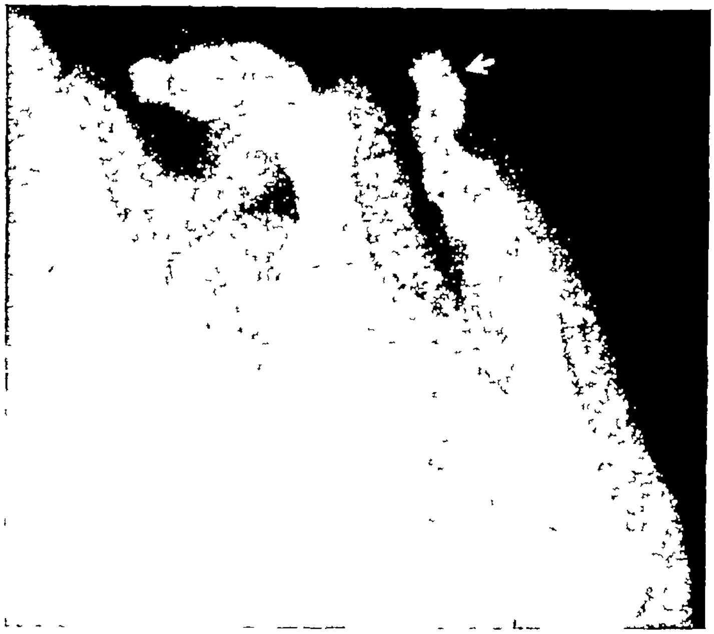


Fig 120 —True uncommon type of subungual exostosis of fifth distal phalanx (Courtesy Dr Frank Weinstein)

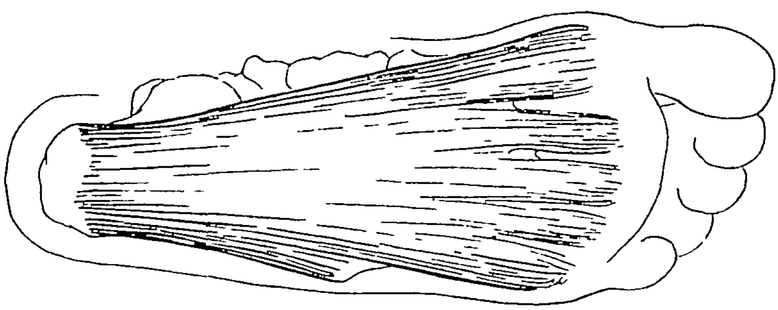
Calcaneal Spur

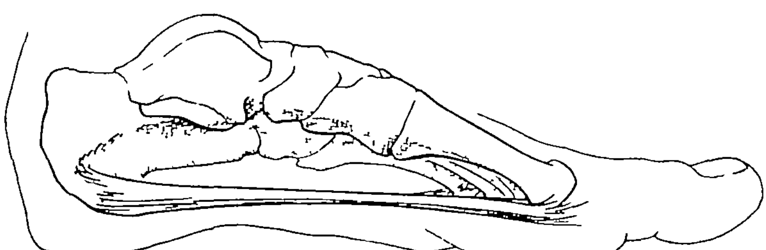
Heel spur is an osteophytic outgrowth just anterior to the tuberosity of the calcaneus, extending along its entire width, for about 2 to 2 5 cm The apex of the spur is embedded in the plantar fascia, directly anterior to its origin The condition may exist without symptoms or it may become painful, even disabling

Calcaneal spurs are of three types (1) those which are large but symptomless, because the angle of growth is such that the spur does not become a weight-bearing point, or those in which the inflammatory changes have been arrested, so that the condition is disclosed only incidentally during roentgenologic examination of the foot made for some other purpose, (2) those which are large and painful on weight bearing, because the pitch of the calcaneus has been altered by a depression of the longitudinal arch, the spur thus becoming a weight-bearing point, (3) those having only a rudimentary proliferation and an irregular, jagged outline accompanied by an area of decreased density around the origin of the plantar fascia, indicating a subacute inflammatory process (Fig 121) All

Fig 121 —Heel spur undergoing subacute inflammation

A





B

Fig 122 —A, Plantar fascia, narrow at origin and wide at insertion B, Plantar fascia as a bowstring (From DuVries, H L A M A Arch Surg 74 536, April, 1957)

calcaneal spurs undoubtedly begin as the third type, but only a few become symptomatic at that stage, because only in those few are the etiologic factors acute

Etiology.—The cause of heel spurs was long obscured, because it was believed that they were due to gonorrheal infection (gonorrheal spurs) As late as 1932, Liberson began his article with, "The painful heel of gonorrhea is due to both bony exostosis and the soft tissue infiltration over it." Later writers suggested infectious diseases and trauma as causes of spur formation Blokhin and Vinogradova (1937) reported thirty-three cases in which arteriosclerosis, gonorrhea, and syphilis were not at all causative The spur resulted from functional overuse or an abnormality of the bones of the foot, or both circumstances. In eighteen cases studied by Costa Bertani (1939), heel spur was associated with flatfoot in 40 per cent of the patients Davis and Blair (1950) reported fifteen cases of spurs of the calcaneous associated with Strumpell-Marie disease The hereditary factor has been suggested by Gould (1942), who reported exostosis of the heel in the father of six children, the third child, a son, had a spur. That son had four children, of whom the second child, a boy, had a heel spur Gould suggested that the abnormality passed down through the male side

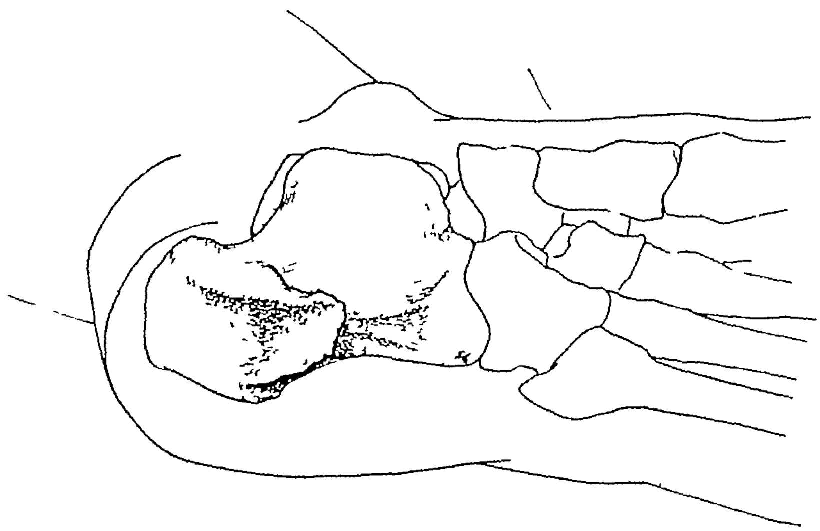


Fig 123 —Spur extends over entire width of tuberosity (From DuVries, H L A M A Arch Surg 74 536, April, 1957)

The important factor in the cause is undoubtedly mechanical rather than infectious and is in all probability the result of the following chain of events The plantar fascia arises from the anterior lip of the tuberosity of the calcaneus, which is about 2 cm wide From this origin it fans out over the entire plantar surface of the foot and is inserted into the fascial plane under the five metatarsal heads, measuring at that point about 10 cm in width (Fig 122) The plantar fascia functions as a bowstring for the entire plantar surface of the foot, thus, any excess strain on the longitudinal arch exerts a maximum pull or strain at the acute angle of its origin A so-called weak foot and overweight, which alter the pitch of the calcaneus, are instrumental in exerting excess strain Over a pro-

longed period, excessive strain produces proliferative bone changes at the origin of the fascia, ultimately forming the spur.

Diagnosis.—The principal symptom is severe pain in the entire plantar surface of the heel, which is aggravated by weight bearing and becomes progressively worse and often incapacitating. On palpation, the entire plantar surface of the heel is tender, but the point of maximum tenderness is elicited just anterior to the calcaneal tuberosity. This tender region may be pinpointed on a roentgenogram immediately under the spur.

In the early stages, fibrositis of low chronicity with or without pain anterior to the calcaneal tuberosity represents the pathologic change. Continuation of the process leads to osteophytic changes and bone deposits in the sulcus, just anterior to the tuberosity. The accumulation of new bone is self-limiting, the final spur varies greatly in size and shape, but mostly it is a triangular bar shape. The usual roentgenographic mediolateral view is two-dimensional, the spur appears pointed like a tack, but actually it extends over the entire width of the tuberosity (Figs. 123 and 124).

Fig. 124.—Medial view of heel spur on a skeleton.

Treatment.—The asymptomatic type need not be treated. Most of the fully formed symptomatic spurs respond to mechanical measures, such as proper footgear, correction of any static foot disability, increased rest of the foot, and a foot appliance having a transverse sulcus in its heel portion to accommodate the spur. Injection of procaine hydrochloride, alcohol, sclerosing agents, or hydrocortisone acetate has been tried, but their therapeutic efficacy is doubtful, because none of the published studies reviewed reports the use of a control group.

Surgical excision of the spur is indicated for the intractable type. Both medical and mechanical measures should be thoroughly tried before surgical intervention is contemplated. At the stage of active fibrositis, palliative measures probably will arrest the disease, surgical intervention may aggravate it.

A

B.

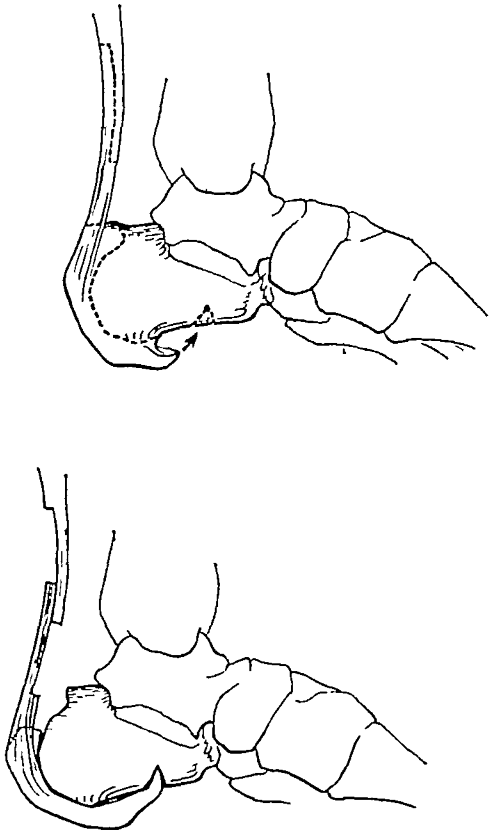


Fig 125 —Steindler's rotation osteotomy for heel spur *A*, Tendo achillis lengthened, osteotomy performed in posterior portion of calcaneus *B*, Posterior portion of calcaneus rotated so that spur fits into previously prepared notch in plantar surface of body of calcaneus (From DuVries, H L A M A Arch Surg 74 536, April, 1957)

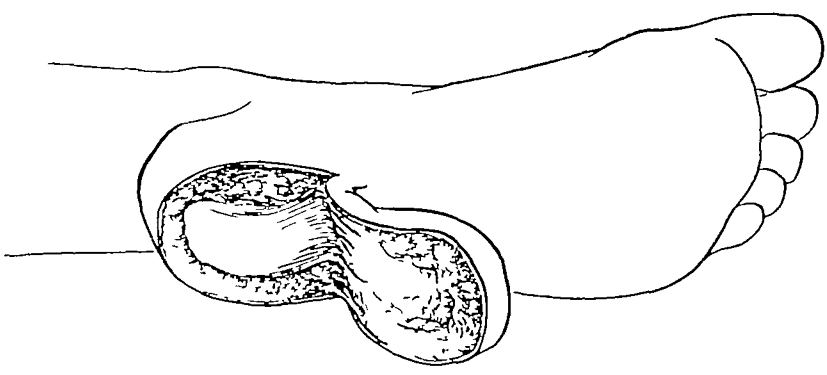


Fig 126 —Griffith's procedure Entire plantar surface of heel denuded as a flap, spur removed, flap sutured into place (From DuVries, H L A M A Arch Surg 74 536, April, 1957)

Several operative procedures have been devised for the treatment of heel spurs Steindler's (1940) rotation osteotomy (Fig 125) of the calcaneus and lengthening of the tendo achillis is designed to take all the weight off the point of the spur by rotating the posterior portion of the calcaneus in such a manner that the spur comes to lie in the horizontal plane and not in a vertical or oblique plane

Griffith's Procedure —Griffith's (1901) procedure, also described by Chang (1934), is widely practiced A U-shaped incision is made completely around the heel, and the skin flap is reflected, exposing the plantar surface of the heel at the origin of the plantar fascia (Fig 126) The fascia is severed at its origin, followed by amputation of the spur

Blokhin's Modified Chang Technique.—Blokhin and Vinogradova (1937) reported thirty-three cases in which they used a modified Chang and Miltner technique successfully in the seventeen patients whom they followed postoperatively for one to four years

All the foregoing procedures are unnecessarily traumatic

Recommended Technique.—In a personal series (1957) of fifty cases in which operation was clearly indicated, the recommended procedure was employed without a single recurrence or residual disability The procedure is comparatively atraumatic, and the resulting scar formation at the origin of the plantar fascia has more tensile strength than the normal anatomic attachment of this fascia, it is therefore able to withstand more stress than normally

1 Make a linear incision over the medial side of the calcaneus about 2 cm above the plantar border (Fig 127) This is usually immediately over the medial border of the plantar fascia Begin the incision at about the posterior border of the calcaneus and continue to about the anterior third of this bone The incision need not be longer than 5 or 6 cm The spur at this step is immediately under and in the middle of the incision

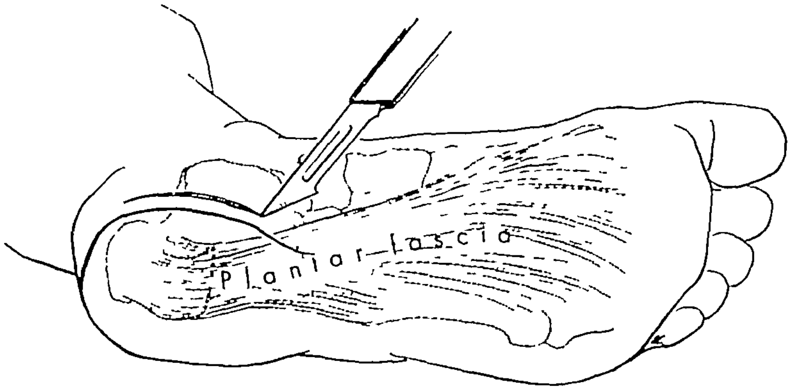
2 Incise in depth until the medial border of the plantar fascia is exposed Place the index finger of the right hand over the superior surface of the plantar fascia just anterior to its origin in an attempt to free it completely across to the lateral side (Fig 128) Dissect with scissors until the fascia is freed over and around the spur and is completely denuded on its superior and inferior surfaces across from the medial to the lateral side (Fig 129)

3 Place the blade of an 8 mm osteotome against the base of the spur, pressure of the palm of the right hand against the osteotome is sufficient to amputate the spur across the entire surface of its base (Fig 130)

4 After excision, smooth the surface with a Joseph nasal rasp (Fig 131)

5 Suture the fascia and skin in layers (Fig 132) and apply a compression bandage Other means of immobilization are not required

6 The patient may assume weight bearing with the aid of crutches in about four days Remove sutures in ten to twelve days In about four weeks postoperatively, full weight bearing may be resumed A sponge-rubber mold, fashioned from a plaster mold of the foot, stabilizes the foot and hastens recovery



Figs 127 to 132 —DuVries' technique

Fig 127 —Linear incision about 6 cm long made on medial side, about 2 cm above medioplantar border of skin (From DuVries, H L A M A Arch Surg 74 536, April, 1957)

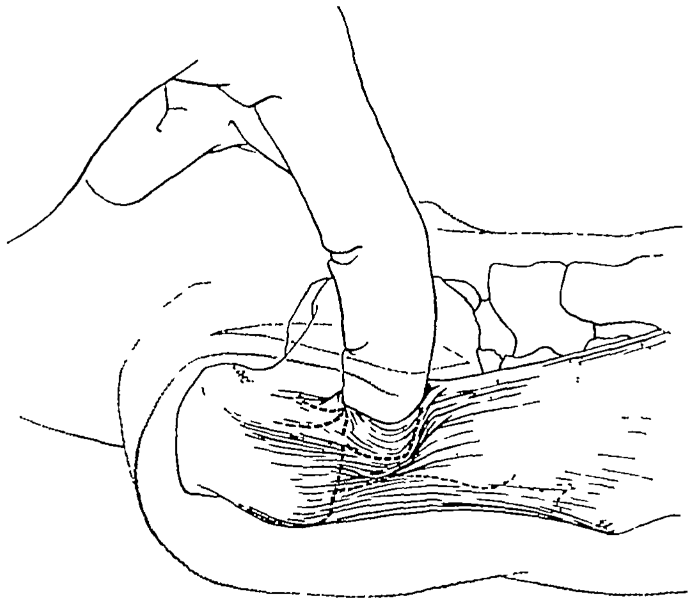


Fig 128 —DuVries' technique Index finger frees space immediately above plantar fascia and apex of spur (From DuVries, H L A M A Arch Surg 74 536, April, 1957)

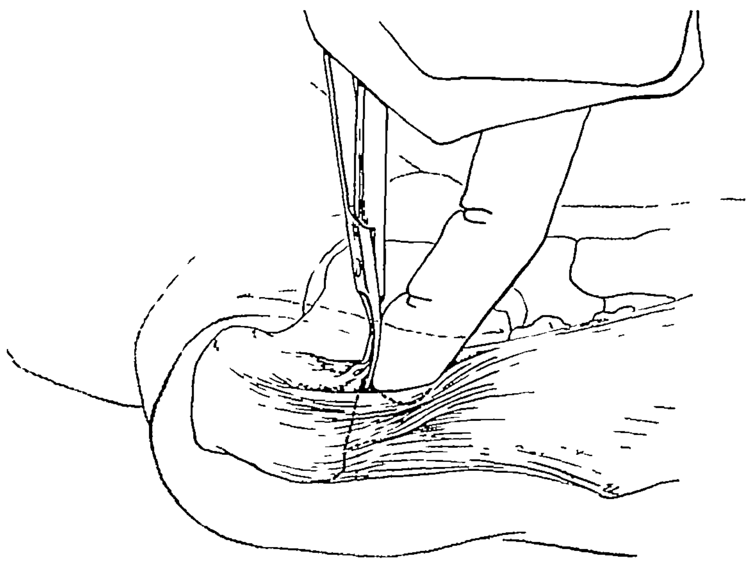


Fig 129 —DuVries' technique With scissors, all integument freed from spur (From DuVries, H L A M A Arch Surg 74 536, April, 1957)

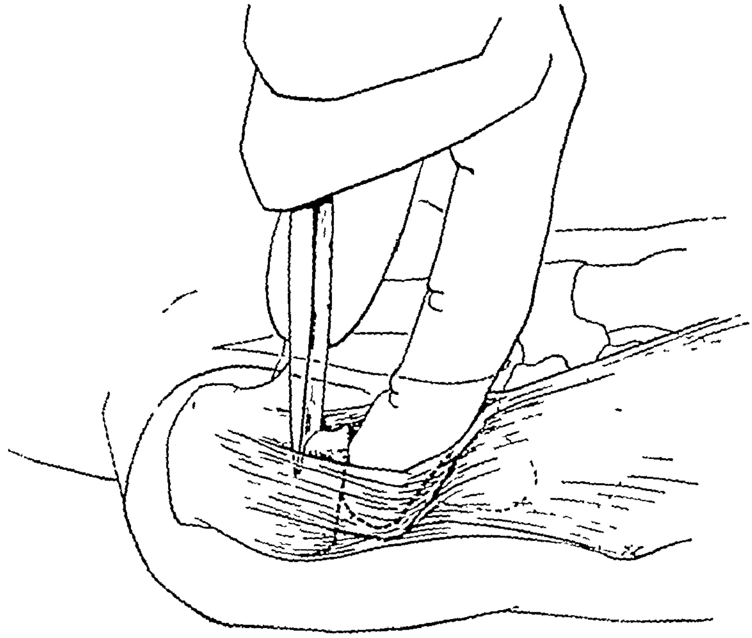


Fig 130 —DuVries' technique With an 8 mm osteotome, spur is amputated (From DuVries, H L A M. A Arch Surg. 74 536, April, 1957)

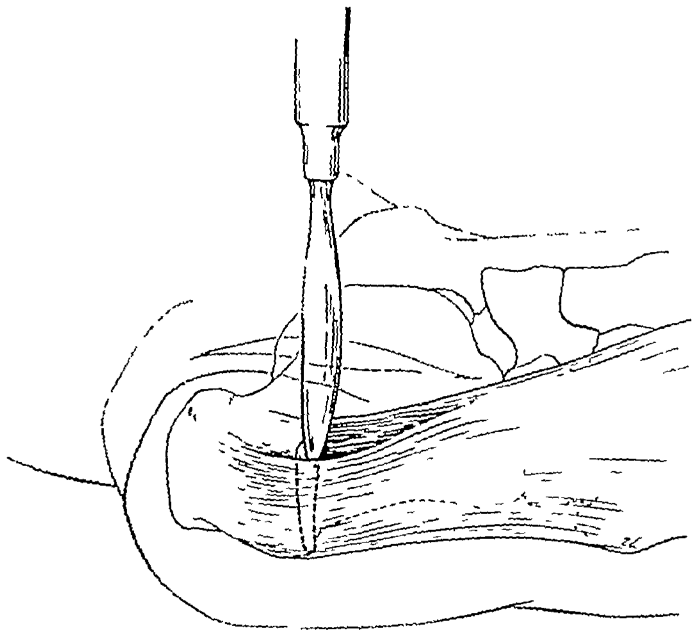


Fig 131 —DuVries' technique Surface of amputation is smoothed with Joseph nasal rasp (From DuVries, H L A M A Arch Surg 74 536, April, 1957)

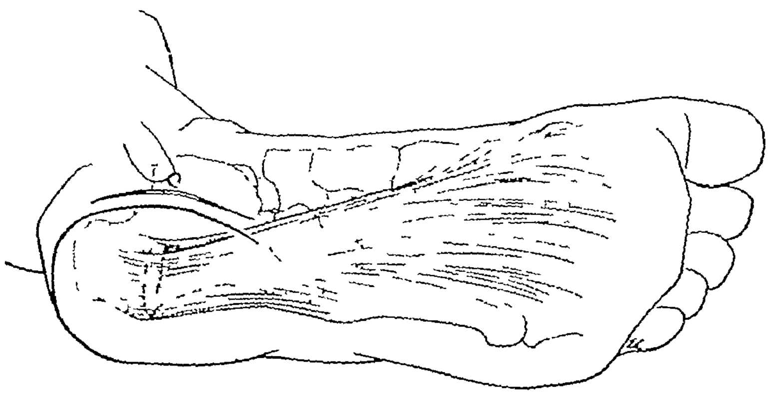


Fig 132 —DuVries' technique Fascia and skin closed in layers (From DuVries, H L. A M A Arch Surg 74.536, April, 1957)

The advisability of surgical removal of spurs has been questioned on the ground that the spur tends to recur. My experience does not coincide with this opinion, and published reports do not support the contention inasmuch as they do not include comparisons of preoperative, immediately postoperative, and late postoperative roentgenograms to demonstrate recurrence of the spur. Certainly, some roughening of the bone at the point of amputation of the spur does form

Fig 133

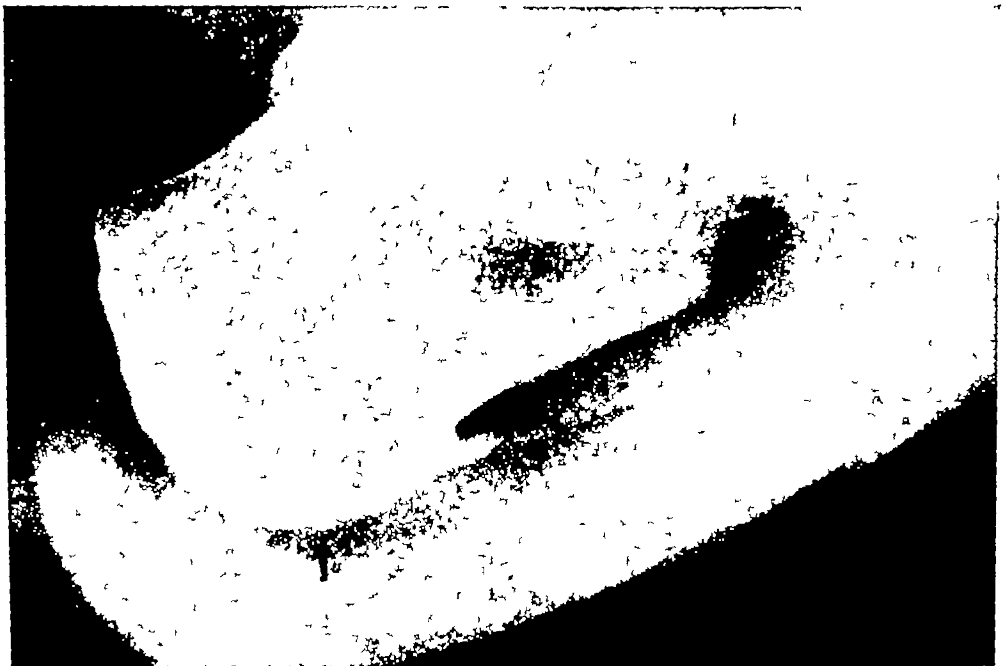
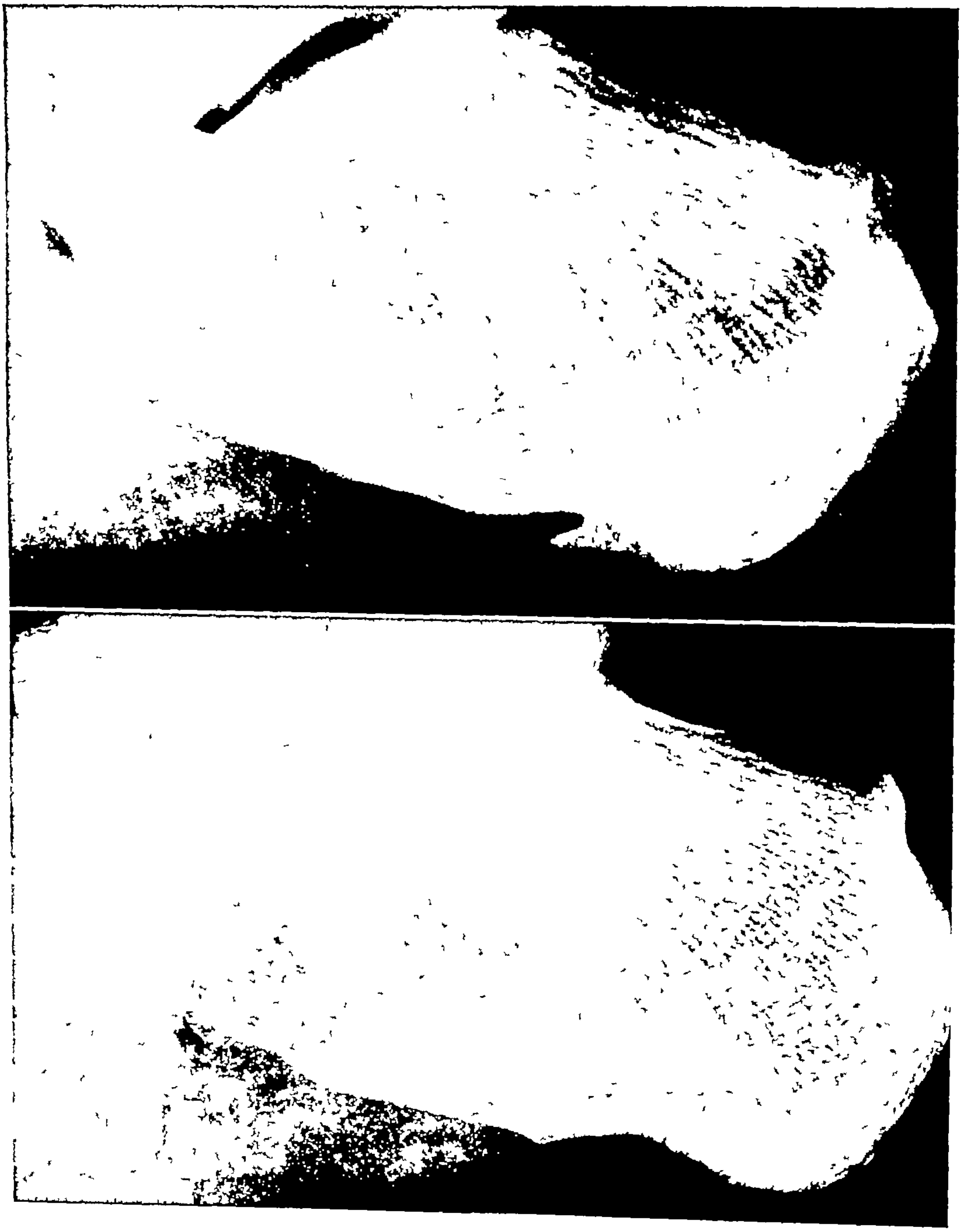


Fig 134

Figs 133 and 134 —Presumably removed heel spur. Note chiseling on plantar tuberosity

in some cases, however, such roughening has not become symptomatic in any personal cases In two in which spurs were said to have been removed earlier elsewhere, the spur was as smooth in outline as any preoperative spur, nevertheless, the plantar tuberosity of the calcaneus was roughened as though it had been chiseled (Figs 133 and 134) Evidently the operator had overlooked the spur and removed a part of the tuberosity, which caused the roughened surface

A.



B

Fig 135 —A, Painful spur, preoperatively B, Three years postoperatively Symptom-free six years postoperatively

The spur which was present could not have been a recurrent spur, for if recurrent, the spur's cortex would be rough I removed the spur in each of these two cases Spurs have not recurred after fifteen years

A

B

Fig 136 —*A*, Painful spur, preoperatively *B*, Three years postoperatively Still symptom-free six years postoperatively

I have seen many patients as long as twenty years after removal of calcaneal spur, all are completely free from painful heel (Figs 135 and 136)

REFERENCES

Abramson, D I Diagnosis and Treatment of Peripheral Vascular Disorders, New York, 1956, Harper & Bros

Ackerman, L V, and del Regato, J A Cancer, ed 3, St Louis, 1954, The C V. Mosby Co

Aird, I, Johnson, H D, Lennox, B, and Stansfeld, A. G · Epithelioma Cuniculatum· Variety of Squamous Carcinoma Peculiar to Foot, Brit J Surg 42 245-250, Nov., 1954.

Allen, A C, and Spitz, S Malignant Melanoma A Clinicopathological Analysis of Criteria for Diagnosis and Prognosis, Cancer 6 1-45, Jan, 1953

Allen, R A, Woolner, L B, and Ghormley, R K Soft Tissue Tumors of Sole, With Special Reference to Plantar Fibromatosis, J Bone & Joint Surg. 37A 14-26, Jan, 1955.

Blokhin, V N, and Vinogradova, T. P Calcaneal Spurs, Ortop i travmatol (no. 1) 11.96-113, 1937

Browne, H J Squamous Carcinomas of the Extremities, Thesis, University of Minnesota, 1953

Castleman, B Presentation of a Case (Ewing's Tumor of the Calcaneum), New England J Med 252 399-402, Mar 10, 1955

Castleman, B Presentation of a Case (Aneurysmal Bone Cyst), New England J Med 253· 75-77, July 14, 1955

Cater, D B Basic Pathology and Morbid Histology, Baltimore, 1953, Williams & Wilkins Co

Chamberlain, J W, and Lawrence, K B Congenital Sarcoma of Extremities, Report of Two Cases, Am J Dis Child 62 793-800, Oct, 1941.

Chang, C C, and Miltner, L J Periostitis of the Os Calcis, J Bone & Joint Surg 16 355-364, Sept, 1934

Charache, H Squamous Cell Carcinoma of Extremities, Surg Gynec. & Obst 68 1002-1006, June, 1939

Child, P L Liposarcoma of the Os Calcis, Am J Clin Path 25 1050-1052, Sept, 1955

Cohen, J, Brown, K A, and Grice, D S Ewing's Tumour of Talus (Astragalus) Simulating Aseptic Necrosis, J Bone & Joint Surg 35A 1008-1012, Oct, 1953

Coley, B L, and Higinbotham, N L Tumors Primary in Bones of Hands and Feet, Surgery 5 112-128, Jan, 1939

Coley, B L Neoplasms of Bone and Related Conditions, New York, 1949, Paul B Hoeber, Inc

Coley, B L, and Higinbotham, N L Tumors of Bone A Roentgenographic Atlas, Annals of Roentgenology, vol 21, New York, 1953, Paul B Hoeber, Inc, pp 100-111

Copeland, M M Tumors of Cartilaginous Origin, Clin Orthoped, No 7, p 9, 1956

Costa Bertani, G Flatfoot as Factor in Producing Inferior Exotosis of Heel, Rev Asoc méd argent 53 455-462, April, 1939

Davis, J B, and Blair, H C Spurs of Calcaneus in Strumpell-Marie Disease Report of 15 Cases, J Bone & Joint Surg 32A 838-840, Oct, 1950

Decker, A M, and Chamness, J T Melanocarcinoma of Plantar Surface of Foot, Review of 2 Cases, Surgery 29 731-742, May, 1951

Durbin, F C, and Smith, G S A Chondromatous Tumour of the Calcaneum, J Bone & Joint Surg 37B 584, 1955

DuVries, H L Heel Spur (Calcaneal Spur), A M A Arch Surg 74 536, April, 1957

Fahey, J J, and Bollinger, J A Congenital Sarcoma of the Foot Case Report and Review of Literature, A M A Am J Dis Child 86 23-27, July, 1953

Geschickter, C F, and Copeland, M M Tumors of Bone, Philadelphia, 1949, J B Lippincott Co

Gould, E A Three Generations of Exostoses of Heel Inherited From Father to Son, J Hered 33 228, June, 1942

Griffith, J D Osteophytes of the Os Calcis, Am J Orthoped Surg 8 501, 1901

Jaffe, H L Aneurysmal Bone Cyst, Bull Hosp Joint Dis 11 3-13, April, 1950

Jaffe, H L Osteogenic Sarcoma of Bone, Clin Orthoped, No 7, p 27, 1956

Jaffe, H L, and Lichtenstein, L Nonosteogenic Fibroma of Bone, Am J Path. 18 205-221, March, 1942

Jaffe, H L, Lichtenstein, L, and Portis, R B Giant Cell Tumor of Bone, Its Pathologic Appearance, Grading, Supposed Variants and Treatment, Arch Path 30 993-1031, Nov, 1940

Johnson, E W, Ghormley, R K, and Dockerty, M B Hemangiomas of the Extremities, Surg Gynec & Obst 102 531-538, May, 1956

King, E S J Malignant Tumours of Tendon Sheaths, Australian & New Zealand J Surg 10 338-353, April, 1941

King, E S J Glomus Tumour, Australian & New Zealand J Surg 23 280-295, May, 1954

Kingsbery, L B Solitary Cyst of the Os Calcis in Adults and Children, Report of Eight Cases, J Internat Coll Surg 32B 83-100, Jan, 1957

Kleinberg, S Angioma of Foot, J Bone & Joint Surg 24 367-371, April, 1942

Kulchar, G V Benign and Malignant Tumors of the Foot, J A M A 124 761-766, March 18, 1944
Liberson, F Deep X-ray Therapy in the Treatment of "Painful Heel," J Urol 28 105-115, July, 1932
Lichtenstein, L Aneurysmal Bone Cyst, J Bone & Joint Surg 39A 873, July, 1957
Lichtenstein, L Bone Tumors, St Louis, 1952, The C V Mosby Co
Lichtenstein, L Aneurysmal Bone Cyst, Further Observations, Cancer 6 1228-1237, Nov, 1953
Lichtenstein, L Tumors of Synovial Joints, Bursae, and Tendon Sheaths, Cancer 8 816-830, July-Aug, 1955
Lichtenstein, L, and Hall, J E Periosteal Chondroma Distinctive Benign Cartilage Tumor, J Bone & Joint Surg 34A 691-697, July, 1952
Lippsett, D H, and Johnson, J R Diffuse Lipomatosis of the Lower Extremity, Report of a Case, Bull Ayer Clin 4 55, 1954
Longstreth, H P, Blanco, P, and Sanes, S Myxochondrosarcoma of Talus, J Bone & Joint Surg 30A 774-778, July, 1948
Meyerding, H W Multiple Metatarsal Fractures Associated With Osteogenic Sarcoma, J A M A 124 228-230, Jan 22, 1944
Miles, J E, and Degenshein, G A Solitary Bone Cyst, Am J Surg 91 170-176, Feb, 1956
Minear, W L Xanthomatous Joint Tumors, J Bone & Joint Surg 33A 451-458, April, 1951
Moberg, E Natural Course of Osteoid Osteoma, J Bone & Joint Surg 33A 166-170, Jan, 1951
Ottley, C M Glomus Tumour, Brit J Surg 29 387-391, April, 1942
Oughterson, A W, and Tennant, R Angiomatous Tumors of Hands and Feet, Surgery 5 73-100, Jan, 1939
Pack, G T Tumors of Hands and Feet, Introduction, Surgery 5 1-26, Jan, 1939
Pack, G T, and Adair, F E Subungual Melanoma, Differential Diagnosis of Tumors of Nail Bed, Surgery 5 47-72, Jan, 1939
Pack, G T, and Ariel, I M Synovial Sarcoma (Malignant Synovioma), Report of 60 Cases, Surgery 28 1047-1084, Dec, 1950
Pack, G T, Lenson, N, and Gerber, D M Regional Distribution of Moles and Melanomas, A M A Arch Surg 65 862-870, Dec, 1952
Pascher, F, and Sims, C F Basal Cell Epitheliomas of Sole, Report of Two Cases, A M A Arch Dermat & Syph 69 475-481, April, 1954
Roaf, R Ewing's Tumour Affecting Medial Cuneiform Bone, J Bone & Joint Surg 34B 640-641, Nov, 1952
Schreiner, B F, and Wehr, W H Primary Malignant Tumors of Foot A Report of 37 Cases, Radiology 21 513-521, Dec, 1933
Steindler, Arthur Orthopedic Operations Indications, Technique, and End Result, Springfield, Ill, 1940, Charles C Thomas, Publisher, p 216
Stout, A P Liposarcoma-Malignant Tumor of Lipoblasts, Ann Surg 119 86-107, Jan, 1944
Stout, A P Tumors of Soft Tissues, Washington, D C, 1953, Armed Forces Institute of Pathology
Warnick, R T, and Thomson, A D Bone Tumors A Histological Method of Classification, Arch Middlesex Hosp 4 219, 1954
Willis, R A Spread of Tumours in the Human Body, St. Louis, 1952, The C V Mosby Co

CHAPTER EIGHT

Disorders of the Skin

DISORDERS OF THE SKIN TO WHICH THE FOOT IS ESPECIALLY SUSCEPTIBLE ARE either excrescences caused by friction and pressure over a bony prominence or diseases of the skin itself Friction or pressure may result in hard or soft corns, calluses, keratoses, suppurating sinuses, ulcers, or fibromatoses. Keloids and hypertrophic scars are usually secondary to healing of lacerations, burns, or surgical wounds. Some diseases of the skin are verrucae, dermatoses, especially tinea infections, epidermal cysts, and the rare diastasis of the fifth toe known as *ainhum*

CORNS AND PLANTAR EXCRESCENCES

Corns and calluses are essentially localized keratoses caused by intermittent pressure from without and solid resistance from within, the outside pressure is produced by the shoe, which is resisted from within by the bones *Hard corn (heloma durum)* forms primarily on the exposed surfaces of the toes and tibial side of the great toe joint, or on the fibular side of the fifth metatarsophalangeal joint *Soft corn (heloma molle)* forms over a condyle of a phalanx between the toes *Callus (tyloma)* may form over or under any bony prominence of the foot but commonly does so under the ball of the foot, the plantar surface of the heel, or the base of the fifth metatarsal The terms *heloma* and *tyloma* may be misleading because they imply that the lesion is neoplastic, whereas the lesion is a reactive proliferation secondary to pressure

Corn (Heloma, Clavus)

A corn is an accumulation of horny layers of skin over a bony prominence

Etiology.—The outline of the bones of the foot is irregular, the bones have numerous projections, especially over the condyles of the heads and bases of

the metatarsals and phalanges The shoe presses on those prominent condylar processes, thus, the soft tissues over these prominences bear the brunt of pressure and friction exerted on the foot by ill-fitting shoes Nature attempts to protect the irritated part by accumulating horny epithelium, but the accumulation raises the elevation of the prominence, crowding the foot still more, so that the shoe increases the pressure on the underlying live tissues Excrescences and numerous other morbid changes are inevitable over such pressured areas

That the prominences under excrescences represent proliferative changes in bone is an erroneous concept Galland (1933) and McElvenny (1940) spoke of *exostosis* under corns A personal study of 5,000 roentgenograms of such cases disclosed that only in rare instances are there cortical changes of bone of the condyle In those rare instances, causation was unrelated to causation of the corn

General Treatment.—Palliative measures, such as reducing the horny accumulation and padding the area to distribute the pressure, give relief in most cases, especially relief to nonweight-bearing areas Excrescences on a weight-bearing surface call for orthopedic appliances as well as corrective shoes Intractable conditions over a nonweight-bearing area respond to excision of the condylar prominence immediately under the excrescence (Rutledge and Green, 1957, Billig, 1956) Those excrescences on a weight-bearing area are likely to be associated with some degree of deformity or anomaly of the involved bone They will be discussed under Intractable Plantar Keratoses, page 184

The presence of a *corn on the lateral side of the fifth toe* is common, because the fifth toe receives the maximum pressure of the curve of the outer border of the forepart of standard shoes The head of the proximal phalanx of the fifth toe is ordinarily the most prominent surface at that point, that is why the corn is nearly always over the fibular condyle of the head of the proximal phalanx

Operative Treatment.—The excrescence disappears in almost all cases after treatment by the following simple procedure

1 Under infiltration anesthesia (0 5 ml of 1 per cent procaine hydrochloride), make an incision over the dorsolateral aspect of the toe, extending it from just proximal to the nail to about the base of the proximal phalanx (Fig 137, *A*) The corn itself should not be incised, because the scar would be exposed to further friction, moreover, callous tissues are devitalized, therefore, healing is poor

2 Retract the lateral margin of the skin and subcutaneous tissue to expose the condylar projection covered by capsule and fascia

3 Carry the line of the skin incision into the capsule, denude the bone of the lateral margin of the capsule and retract with the skin (Fig 137, *B*)

4 The condyle has been exposed and may be easily excised (Fig 137, *C*)

5. Smooth the surface with a small rasp

6 Close the skin and capsule with a single mattress suture (Fig 137, *D*)

Postoperative pain is slight Healing is ordinarily rapid, and ambulation may begin in twenty-four hours The toe need never be deformed by this operation

In cases comprising the 2 per cent incidence of recurrence, the head of the proximal phalanx may be amputated, or the middle phalanx may be excised as an alternative procedure, depending on which structure lies under the corn. Excision of the middle phalanx is commonly practiced as the initial operation to remove corns on the fifth toe, but neither procedure is advisable initially because of frequent deforming of the toe and occasional resulting flail toe (Figs. 138 and 139), which sometimes makes amputation of the entire toe necessary.

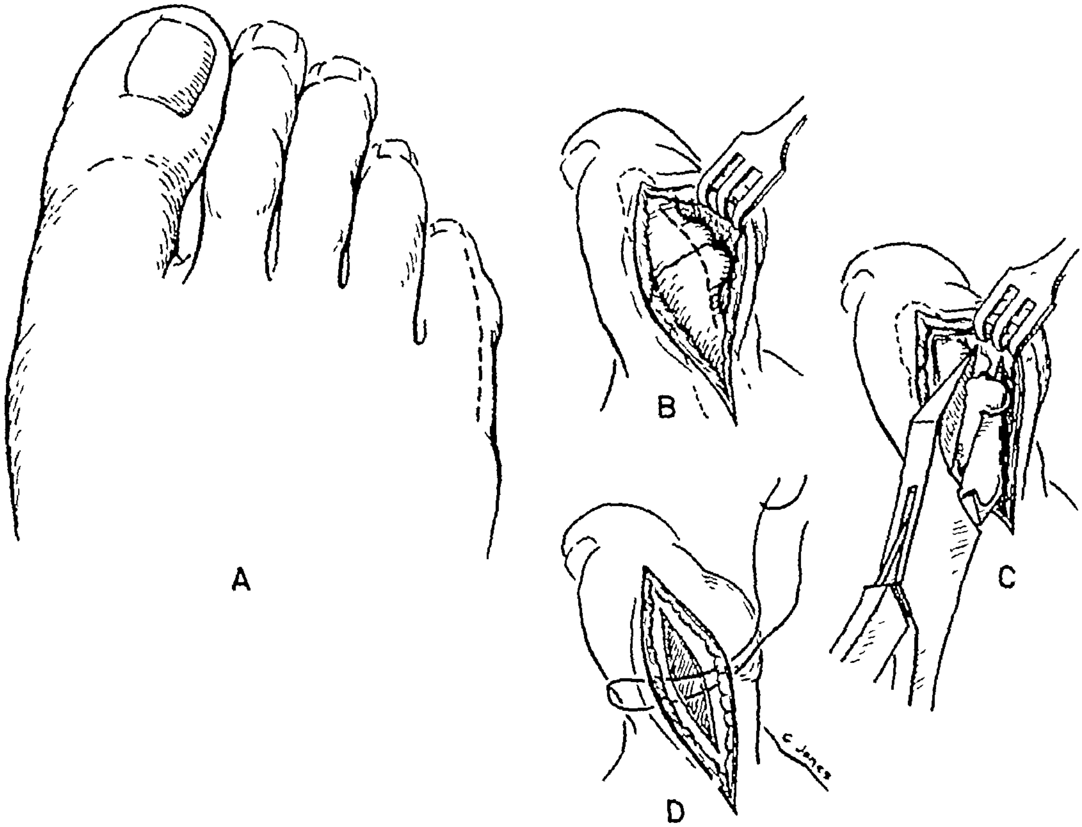


Fig 137 —DuVries' technique for condylectomy of corn on fifth toe *A*, Longitudinal incision over dorsolateral aspect of fifth toe *B*, Skin and capsule retracted *C*, Fibular condyles of phalanges amputated *D*, Skin and capsule closed by mattress suture

Excision of Head of Fifth Proximal or Middle Phalanx —The technique is as follows·

1 Make a horizontal elliptical incision on the outer surface of the fifth toe, completely surrounding the corn and penetrating to the bone

2 Remove all soft structures within the boundary of the incision

3 Expose and amputate the bony prominence That prominence is either the head of the proximal or the body of the middle phalanx

4 Underscore and suture both skin margins

Removal of bone permits coaptation of the skin without tension. The incision heals by first intention in two or three weeks.

Corns on the dorsum of the middle three toes and the great toe are for the most part secondary to *hammertoe* (See pages 347 to 356)

Soft corns in the web between the fourth and fifth toes are due to long-continued compression of the skin between the head of the fifth proximal

Fig 138

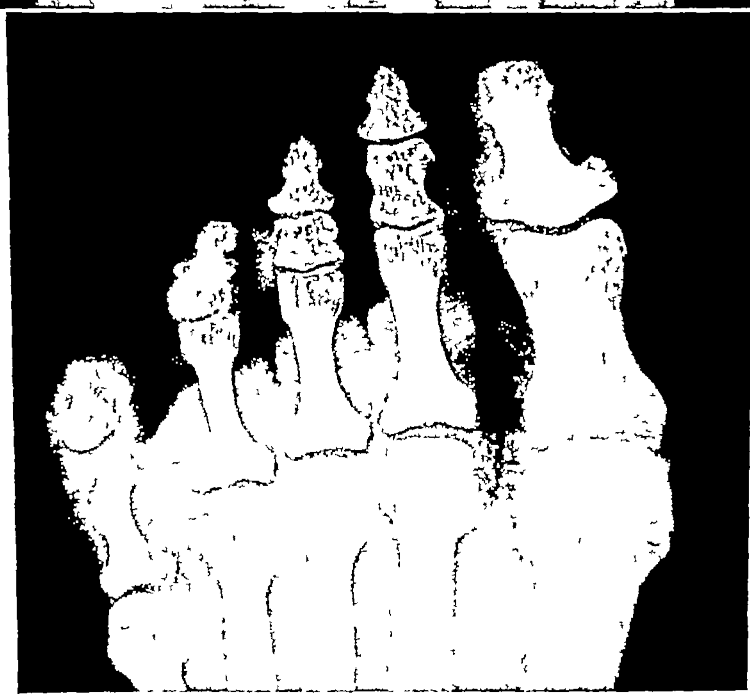


Fig 139

Figs 138 and 139 —Flail toes produced by phalangectomy

Fig 140 —Right foot, head of fifth proximal phalanx adjacent to base of fourth proximal phalanx impinges on soft tissue in fourth web, causing a severe soft corn Left foot same patient, no impingement Sharp point on tibial side of head of right first metatarsal frequent cause of sinus formation over it (See Fig 144)

phalanx and the base of the fourth proximal phalanx (Fig 140) They comprise a common disability A sinus leading into the metatarsal interspace with recurrent episodes of acute infection often complicates this lesion.

Treatment by Condylectomy—Condylectomy of the tibial side of the head of the fifth proximal phalanx gives relief The procedure is similar to that described for *hard corn of the fifth toe,* except that the incision is made on the dorsomedial aspect of this toe Amputation of the condyle on the fibular side of the base of the fourth proximal phalanx also gives relief (Fig 141) This is a more traumatic procedure which is outlined herewith, it is only occasionally indicated

1 Make an incision over the dorsolateral aspect of the fourth proximal phalanx, extending from the middle of the shaft of this phalanx to a point just proximal to the head of the fourth metatarsal (Fig 142,*A*)

2 Open and retract the capsule of the metatarsophalangeal joint to expose the offending condyle for removal by a nasal saw (Fig 142,*B*).

3 Close the skin and capsule by a single layer of sutures (Fig 142,*C*).

Soft corn on any lesser toe may form over any condylar process of the phalanges Condylectomy immediately under the excrescence by a procedure similar to that described for a corn of the fifth toe is effective

Elaborate procedures have been described, but they are all mutilating and are not physiologic Such a technique, for example, is described by Haboush and Martin (1947) who performed a skin plastic operation for soft corn in the fourth toe web (Fig 143) After excision of the soft corn on the fifth toe, a

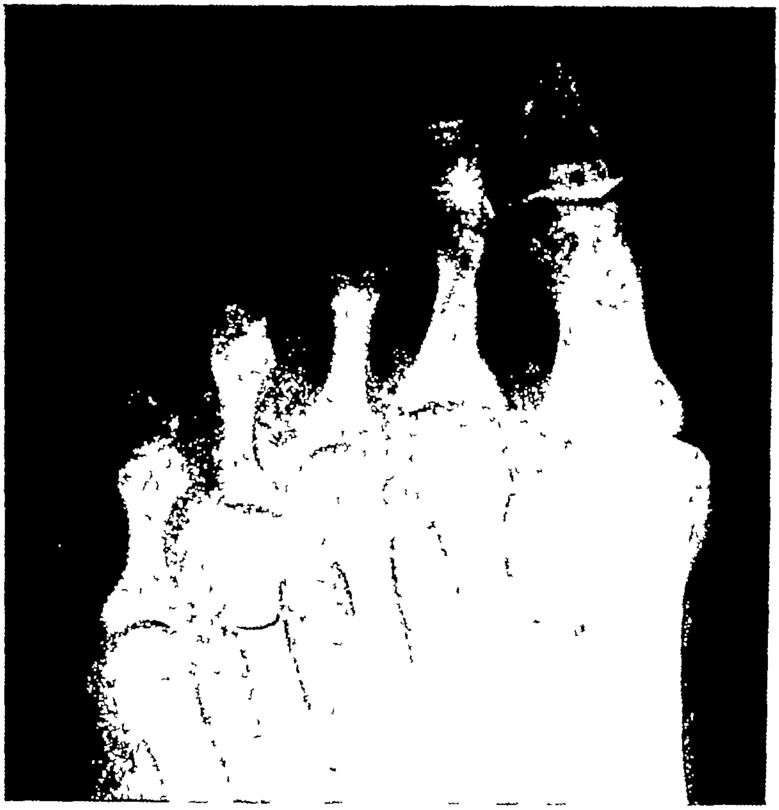


Fig 141 —Postoperative appearance of amputation of condyle of base of fourth proximal phalanx

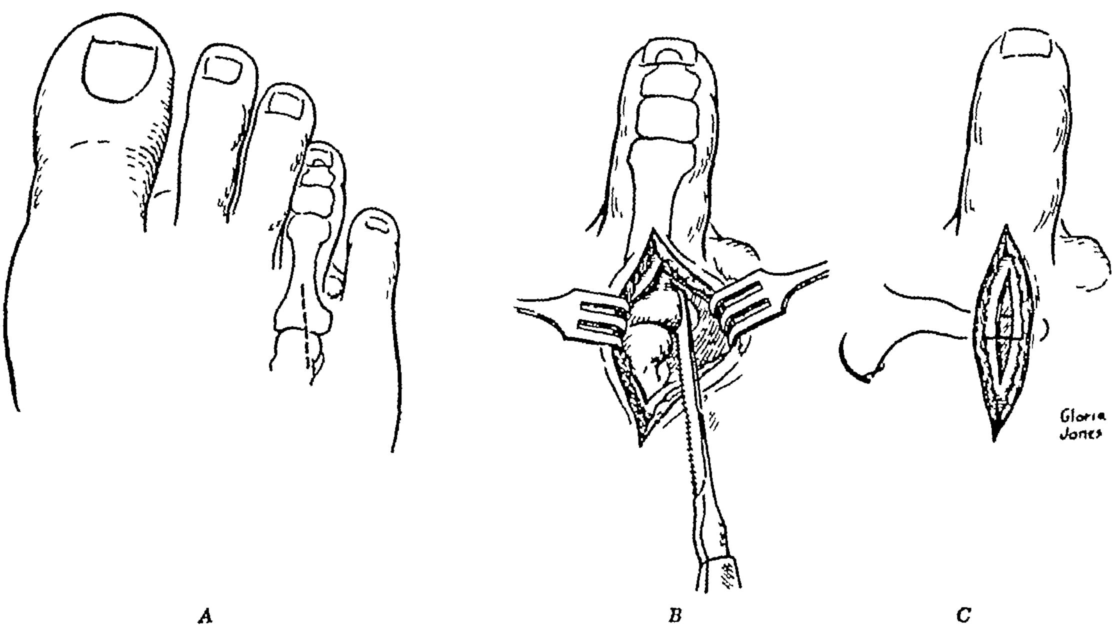


Fig 142 —Technique for excision of base of fourth proximal phalanx for soft corn in fourth web *A*, Incision over lateral aspect of fourth metatarsophalangeal joint *B*, Skin and capsule retracted, fibular condyle of base of fourth proximal phalanx excised with nasal saw *C*, Skin and capsule closed with mattress suture

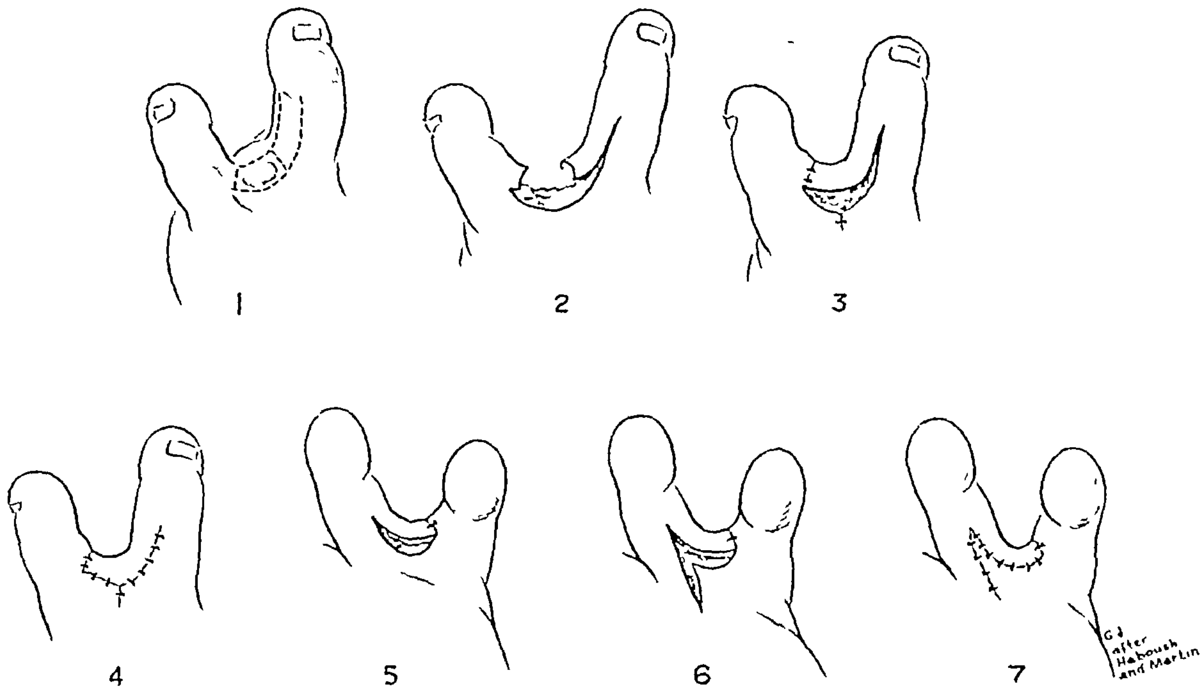


Fig 143 —Haboush and Martin's technique for excision of soft corn *1*, Incisions, *2*, corn excised and flap mobilized, *3*, flap sutured and web advanced, *4*, dorsal closure, *5*, plantar aspect with remaining defect, *6*, flap made and raised, *7*, final suturing

Fig 144 —Probe leading into sinus under corn over tibial side of first metatarsal head, coursing along the head and neck of the metatarsal

skin flap is turned up on the lateral aspect of the fourth toe, based distally, and sutured to the distal edge of the skin from where the corn has been removed The free dorsal edges of the wound on the fourth and fifth toes are then sutured together On the sole of the foot, from the base of the fifth toe in the lateral angle of the still open plantar wound, an incision is made, ¾ inch long, extending proximally The skin flap is then undermined and mobilized, thus effecting a closure of the plantar defect

Corn over the tibial side of the great toe joint ordinarily is secondary to hallux valgus or to an abnormally wide first metatarsal head Such corns often have a degenerative sinus which penetrates to an osseous projection on the tibial side of the head of the first metatarsal (Fig 144)

In mild cases, protective padding and proper fitting of shoes relieve the condition In protracted cases, open reduction of the hallux valgus is necessary for relief In a few cases without valgus deformity in which the condition is due to a congenitally wide head of the first metatarsal segment, a simple condylectomy on the tibial side of the first metatarsal head suffices (For technique, see Bunions, pages 372 to 388)

Corn over the base of the fifth metatarsal is due to an unusual prominence of this bone In most cases, correction is possible by shielding the part by padding and wearing shoes that do not exert pressure over the area In severe cases, reduction of the bony prominence is indicated

Technique for Reduction—The following steps accomplished reduction

1 Make a linear incision over the dorsolateral aspect of the base of the fifth metatarsal, extending from the proximal third of the fifth metatarsal to the lateral aspect of the calcaneocuboid articulation

2 Retract the skin and incise down into the fascia

3 Denude the base of the fifth metatarsal from the lateral margin of the fascia, which includes the insertion of the peroneus brevis tendon, taking care to preserve the peroneal tendon

4 With an osteotome or nasal saw, amputate the lateral prominence of the base of the fifth metatarsal

5 Repair the tendon and fascia with fine chromic catgut, and close the skin in the usual manner

6 Apply a compression bandage which may be released in twenty-four hours The patient may bear weight in forty-eight hours, provided an Oxford is worn which has been cut out over the area of operation

Excrescences on Plantar Surface

The various types of excrescences appearing on the plantar surface are (1) callus, or common tyloma, (2) small, deep-seated, circumscribed nucleated tyloma, (3) verrucae plantaris, solitary or multiple, (4) circumscribed fungating areas due to tinea, (5) epidermal cysts, and (6) intractable plantar keratosis The last five conditions are frequently misdiagnosed and treated as if they were verrucae plantaris

Callus (Tyloma).—Calluses are large keratotic masses histologically similar to helomas but form on the plantar surface of the foot They are due to ex-

cessive pressure on such a weight-bearing surface as the metatarsal heads, heel, or the plantar surface of the base of the fifth metatarsal or under the phalangeal joint of the great toe In severe cases of flatfoot they may occur under the navicular and first cuneiform Thus, again, they are essentially caused by an unequal and pressured weight-bearing surface imposed by ill-fitting footwear

Callus under the first metatarsal head is a circumscribed keratotic area whether or not in association with a hammered great toe. It is formed by weight bearing on a pointed area of a malformed or displaced tibial sesamoid or, occasionally, a fibular sesamoid The keratosis often resembles verrucae plantaris, for which it is mistakenly treated until the keratosis breaks down into a sinus or an ulcer

In mild cases, proper redistribution of weight bearing by means of a shoe inlay or Thomas bar relieves the lesion, in severe cases, reduction of the *hammered toe* or excision of the offending sesamoid or both procedures are necessary (For surgical techniques, see Hammertoe, pages 349 to 356, and Intractable Keratosis, pages 184 to 196)

Callus under the middle three metatarsals is secondary to a depression of the anterior arch, often accompanied by contracted or hammered toes (clawtoes), it is most pronounced in congenital clawfoot.

Etiology—Callus is basically due to short shoes which force the toes to buckle and thus produce a hammertoe deformity (page 348) at the metatarsophalangeal joints Normally, the dorsal angle of the metatarsophalangeal joint is about 160 degrees, in cases of hammertoe the angle may be reduced to 90 degrees, at which angle the base of the proximal phalanx articulates with the dorsum of the head of the metatarsal The pressure of the shoe on the head of the proximal phalanx is transmitted to the metatarsal head which is depressed plantarwise.

Treatment—A metatarsal inlay or Thomas bar added to a properly fitted shoe corrects most mild cases, however, if the lesser toes are greatly contracted, a dorsal tenotomy and capsulotomy of the middle three metatarsophalangeal joints are advisable The toes are then maintained in a plantar overcorrected position for about five weeks If any of the toes are extremely hammered, that deformity should also be reduced (page 354)

Callus under the fifth metatarsal head is generally due to faulty weight distribution of the foot which forces the fifth metatarsal to bear an excessive amount of total body weight Sometimes the callus is caused by an unusually pointed plantar condyle of the metatarsal head

Palliative Treatment—Proper weight redistribution by means of an appliance and well-fitting footwear gives relief In resistant cases, freeing the skin under the metatarsal head or a plantar condylectomy is indicated Sometimes both procedures are necessary The technique is described under Intractable Keratosis (page 184)

Callus on the fibular side of the fifth metatarsal head occurs because the head of the fifth metatarsal is the most prominent point on the outer border of the forefoot The prominence, known as *tailor's bunion*, results from either an

extraordinarily wide fifth metatarsal head or an outward bending of that head. Constant pressure over the prominence often produces keratotic changes. The skin covering it is thin, so that pressure of the shoe keeps it constantly exsanguinated In cold climates, frequent chilling destroys the capillary beds in the skin and subcutaneous tissues, so that the condition is complicated by painful chilblains (Treatment is discussed under Tailor's Bunion, page 399, and Chilblains, page 103)

Callus under the heel is usually due to faulty mechanics of the foot, such as pes planus or pes cavus In either instance, the calcaneus is rotated, so that it bears more weight on one side or the other of the tuberosity In rare instances, the callosity is due to an anomalous shape of the tuberosity, in which part of it projects higher than the rest of the weight-bearing surface

In most cases, correction of the mechanical fault by appliances and proper shoes eliminates the callus In rare cases in which the condition is due to a bony projection of the tuberosity of the calcaneus, the prominent area must be reduced and the surface of the tuberosity leveled The surgical approach to such a prominence can be made from either side of the calcaneus, depending on the site of the prominence

Verruca Plantaris (Papilloma Wart) —Verruca plantaris does not produce the high incidence of disability erroneously attributed to it, because other lesions on the weight-bearing surfaces of the foot assume the appearance of this innocent neoplasm (See Intractable Plantar Keratosis, page 184) The accepted method of treating warts is to destroy them by fulguration, escharotics, excision, or any other means of eradication Lesions that are not verrucae but resemble them *(pseudoverrucae)*, when treated in the manner listed, become intractable Histologically, verrucae plantaris are similar to verrucae vulgaris, grossly they differ largely in that they are embedded below the skin by pressure of weight bearing They differ entirely microscopically from deep-seated keratoses (Fig 145) Warts may occur anywhere on the plantar surface of the foot but only occasionally form under weight-bearing points of pressure, such as under the metatarsal heads or the tuberosity of the calcaneus

Etiology —It is generally accepted that warts are due to a filtrable virus infection They often spread from one part of the body to another A student may acquire a wart and then many in the same classroom may exhibit the growth Kile (1956) concluded that some strains of wart virus are more communicable than others

Diagnosis —It is important to keep in mind that warts, as a rule, do not occur on a weight-bearing prominence, such as a metatarsal head or the tuberosity of the heel bone To rule out verrucae under such surfaces, pinpoint the lesion on a roentgenogram of the foot, if the lesion lies immediately under a weight-bearing point, then differentiate between a neoplasm and a reactive keratosis Typical verrucae are unequivocally circumscribed, either oval or circular in outline, and are completely encapsulated Margins are sharply defined (Fig 146), the center is spongy, pale, and furrowed

Treatment —There are numerous methods of destroying verrucae Many dermatologists and roentgenologists advise radiation therapy, Reeves and Jack-

A

B

Fig 145—*A*, Microscopic section of verrucae Verruca here reveals typical mushrooming of entire epidermis Thickening of rete pegs with some fusion at base and degeneration at top *B*, Hyperkeratosis Hyperkeratinization of uppermost layers of epidermis Rete malpighii, flushed

son (1956) believe that to be the treatment of choice An editorial in the *New England Journal of Medicine* (1953) gives an excellent summary of the treatment of warts by autosuggestion Allington (1952) also discusses psychotherapy of warts, and Dunbar (1947) reviews their psychosomatic origin Duthie and McCallum (1951) advise the use of elastoplast and podophyllin Ducourtioux (1950) suggests electrocoagulation Blank (1947) outlines the following procedure (1) reduce the overlying callus from the verruca, (2) apply a drop of 90 per cent phenol, (3) apply a drop of fuming nitric acid on top of the phenol *The Journal of the National Association of Chiropodists* devoted an entire issue (1954) to discussion of verrucae plantaris Ignatoff (1954) sums up the status of treatment of verrucae plantaris and suggests that once the diagnosis has been established, any of the following procedures will eradicate the growth (1) application of caustics, (2) cauterization, (3) surgical excision, (4) radiation therapy, (5) application of solidified carbon dioxide

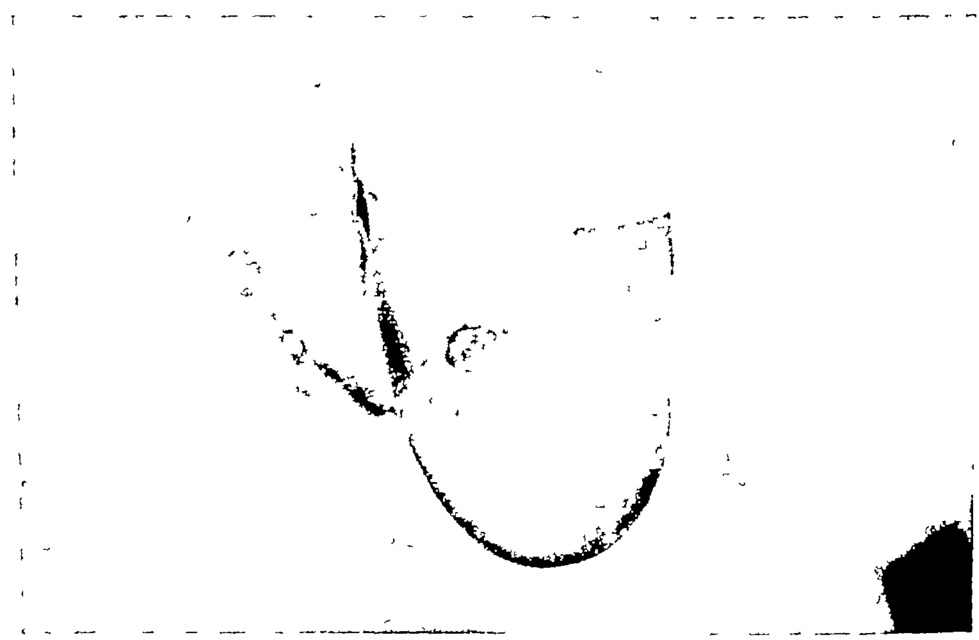


Fig 146 —Typical verruca plantaris on heel

Recommended Technique for Removal of Plantar Wart (Verruca Plantaris) — A drug must be injected, as for an intradermal injection, into the body of the tumor Necrosis of growth results in part from the pressure strangulating the blood supply to the wart and from the sclerosing action of the drug The treatment fails if the needle pierces the capsule, because the solution is rapidly absorbed by the surrounding tissue

1 Inject from 2 to 5 minims of a mild sclerosing material, such as a 3 per cent solution of bismuth sodium tartrate or sodium morrhuate

2 Dress with an aperture pad, and cover the aperture with gauze, felt, or sponge rubber Change the dressing every five to eight days In about a month, 95 per cent of the cases show a scab covering the area which, when removed, discloses healthy skin

3 In 5 per cent of the cases, a residual wart remains The original procedure is then repeated.

4 For the 2 per cent that recur after a second treatment, one may resort to fulguration or excision. Fulguration is best performed by injecting 0 2 ml procaine hydrochloride into the body of the lesion, and then fulgurating the entire mass, including a minute area of normal tissue surrounding the growth

5 To excise verruca plantaris (a) Under general or block anesthesia, make a longitudinal spindle-shaped incision along the entire growth The incision should be long enough so that the margins will coaptate without puckering at the ends (b) Excise all tissue within the incision and remove all subcutaneous

Fig 147 —Excision of large verruca on medial border of heel Skin margins held in apposition by button-retention sutures

fat under the excised area until the fascial plane has been reached (c) Underscore the skin margins and insert one or two retention sutures held by buttons (Fig 147), then suture the skin margins (This procedure may also be used as a secondary procedure for intractable plantar keratosis under the middle metatarsal heads in the small group of recurrences following plantar condylectomy) (d) Apply a compression bandage, which is kept in place for twenty-four hours (e) In forty-eight hours the patient may be ambulatory, provided he wears a cut-out shoe and has an extra heavy layer of gauze dressing over the wound (f) The skin

margin sutures are removed in eight to ten days and the retention sutures in ten to twelve days

Mosaic Plantar Wart.—Typically, the mosaic wart appears on the heel but may form on any other part of the plantar surface, including the web of the toes Those warts extend over a wide area and seem to be a coalescence of multiple small warts They are rather superficial and are relatively painless Montgomery and Montgomery (1937) first called attention to the lesion as a distinct entity Mosaic warts are radioresistant (Montgomery and Montgomery, 1944, 1949) and unresponsive to the usual treatment of warts The Montgomerys (1948) reported 89 9 per cent cures in 109 cases by the following procedure, which is recommended here

The area is pared until the capillary tips are visible or bleeding Capillary bleeding is stopped with silver nitrate solution The wart is swabbed lightly with saturated solution of monochloracetic acid or 20 per cent silver nitrate solution Salicylic acid plaster, shaped to the size of the warty patch, is applied The same treatment is given to the satellites

For hornier lesions, the plaster is fortified with a thin coating of 60 per cent salicylic acid ointment For a deeper reaction, one may rub in a saturated solution of monochloracetic acid instead of silver nitrate solution The plaster is held in place with adhesive strapping The area is kept dry during treatment The lesion is pared down weekly to active warty tissue and the aforementioned steps are repeated When the area thins, silver nitrate solution alone is used Every vestige of wart must be removed to prevent recurrence

When reaction to the application of monochloracetic acid is severe, the medication is discontinued and a wet dressing applied The tissue is pared down to release the subwart fluid Foam-rubber pads, placed posterior to the wart, relieve pressure

Intractable Lesions Under Metatarsal Heads.—Most cases of so-called intractable verruca plantaris under the middle three metatarsal heads are essentially a reactive keratosis resulting from bearing weight on a pointed condylar process under the head of those metatarsals (DuVries, 1953, 1954) Keratotic lesions on the plantar surface, especially those under the heads of the metatarsals or any weight-bearing area of the foot, often become disabling A deep-seated callus or residual scar, when located under a weight-bearing area, can make standing or walking extremely painful Small deep keratotic masses are common under any of the metatarsal heads, especially under the middle three where they are most likely to become intractable, however, a comparable problem occurs under the first metatarsal head, although the anatomic factors differ Under the fifth metatarsal head the condition only occasionally becomes intractable. The degree of disability that these growths may cause varies from a constant discomfort to complete inability to bear weight on the area

Intractable Keratosis Under Middle Three Metatarsal Heads.—The plantar surface of the head of each of the lesser metatarsals has two condylar projections The condylar projection on the fibular side is always the larger of the two Those of the middle three metatarsals end in sharp points extending proximally (Fig 148) The middle three metatarsals form the dome of the

anterior metatarsal arch which functions as a shock absorber and was meant to bear weight only briefly during each step Almost everyone in civilized life has a depression of the anterior arch, therefore, the plantar condyles of the metatarsals almost always bear the full weight As a result, the condylar points gouge the soft structures underneath.

The condylar projections vary in size and shape from one metatarsal to another and in different persons, from a mere rudiment to a projection higher than any other weight-bearing point under all the metatarsal heads, they vary from a ball-like surface to a pin point The sharper and more projecting they are, the more the skin and subcutaneous tissue are subject to fibrotic changes, especially if the fat pad is thin The skin under such a condyle accumulates horny cells as a protective measure The greatei the accumulation of horny

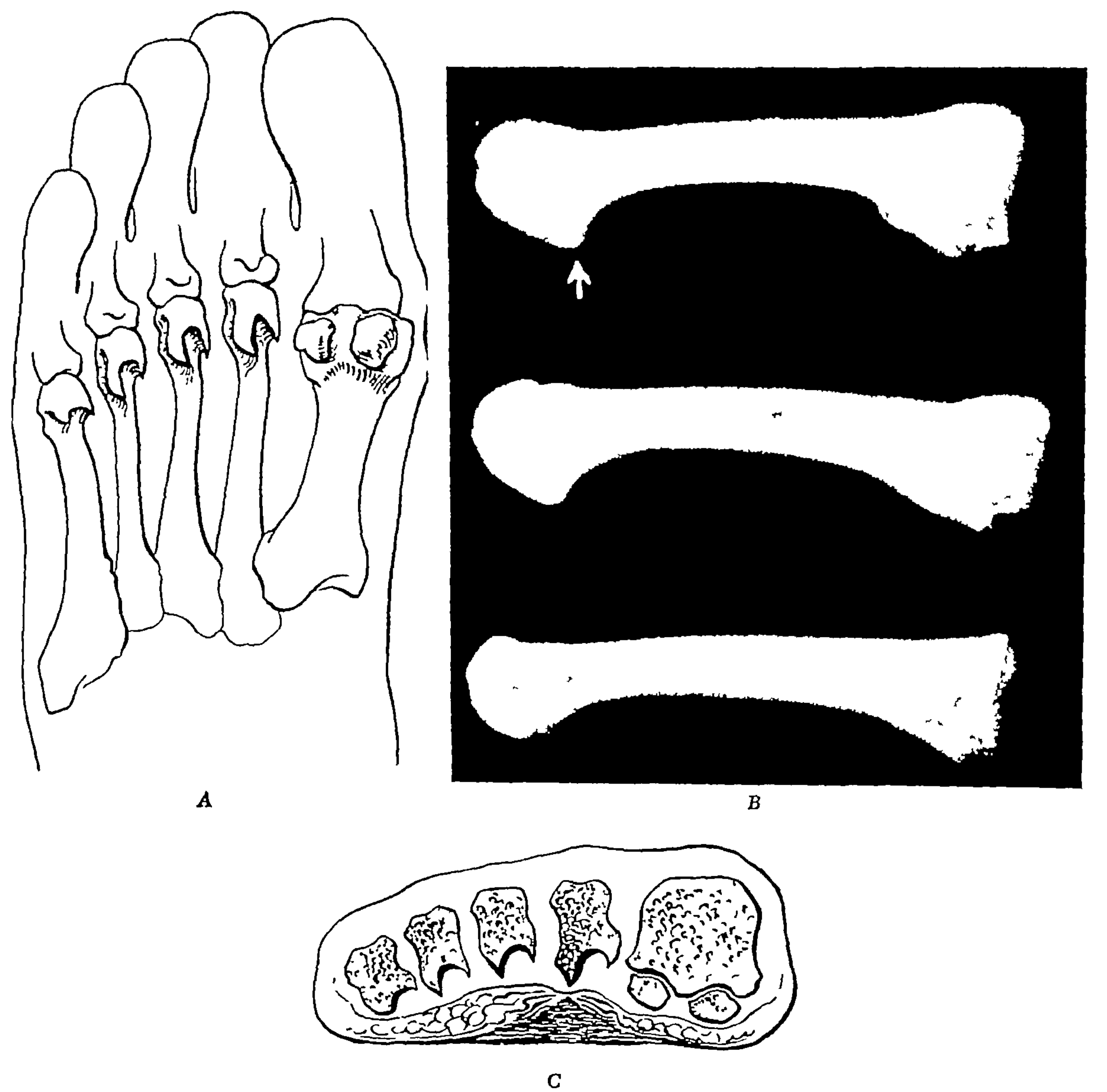


Fig 148—A, Plantar view of sharp condylar process of three middle metatarsals B, Side view of three middle metatarsals Note sharp condylar process C, Cross section of metatarsal heads Hyperkeratosis under sharp condylar process of second metatarsal head

layers, the less space there is between the condylar surface and the contact area, thus, further compression of the soft tissues covering those areas results, and further displacement takes place of the epithelium of the skin and subcutaneous tissue by fibrous connective tissue The fibrous connective tissue which invades the stratum corneum tends to become frayed from friction and appears cauliflower-like (Fig 149) It resembles verrucae, so that when the lesion is mistakenly treated, the scar tissue increases If the erroneous treatment is repeated, as it often is, the lesion becomes intractable

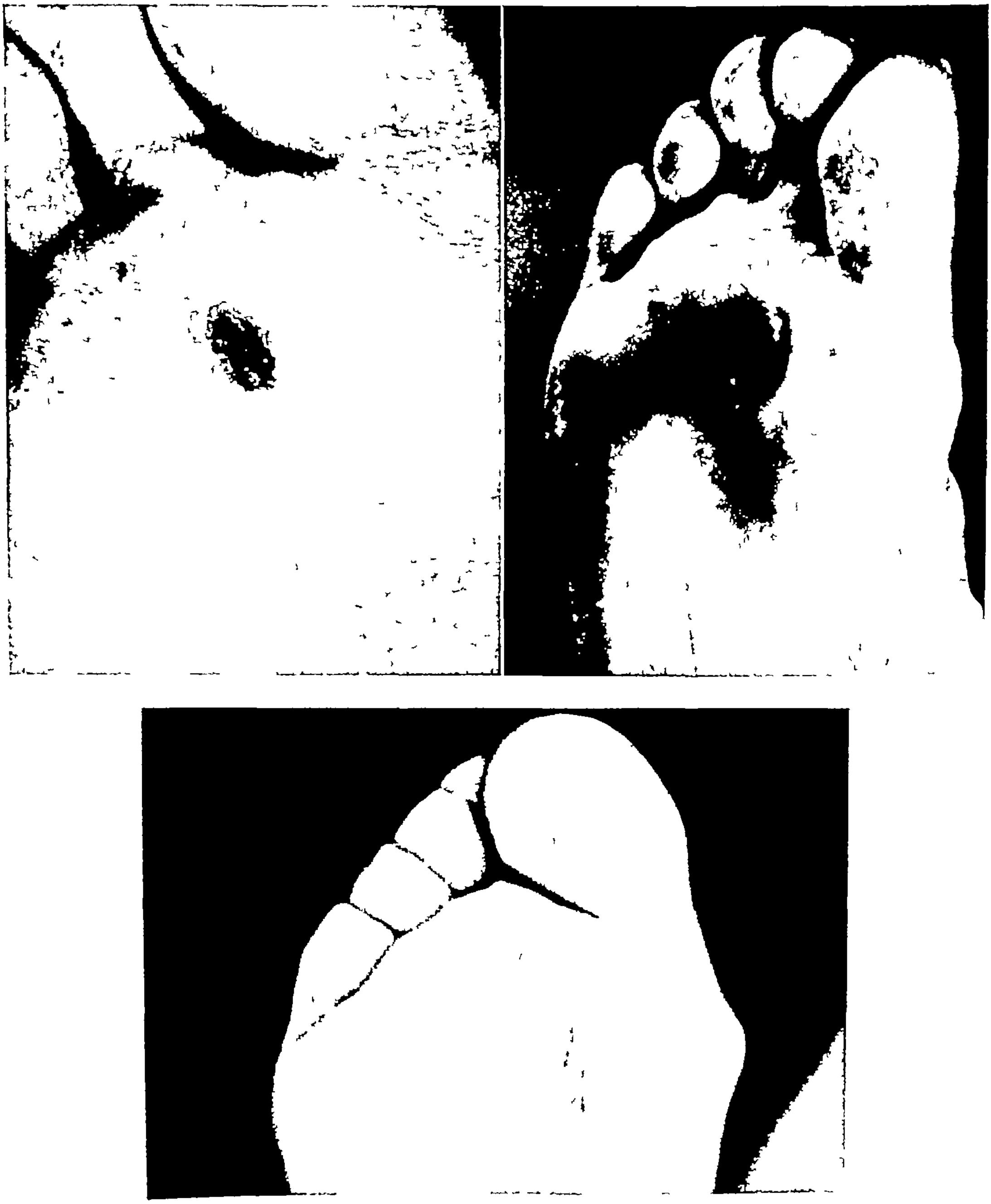


Fig 149 –Different types of plantar keratosis under metatarsal heads, resembling verruca plantaris

Many patients with intractable keratosis become apprehensive of any new treatment for recurrent so-called plantar warts because of their experience with treatment by escharotics, fulguration, excision, and radiation, after which the scar and disability increased The Montgomerys (1949) called attention to fre-

Fig 150 —Increased disability after Dickson's pie technique for intractable keratosis

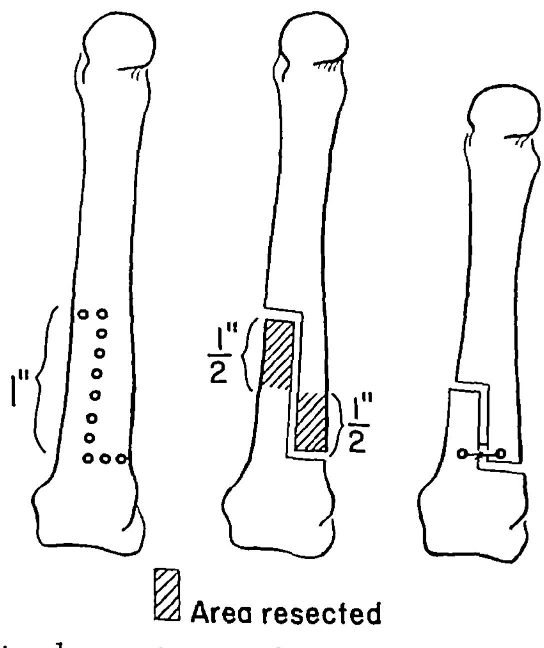


Fig 151 —Giannestra's step-down osteotomy for shortening metatarsal to alleviate plantar keratosis

layers, the less space there is between the condylar surface and the contact area, thus, further compression of the soft tissues covering those areas results, and further displacement takes place of the epithelium of the skin and subcutaneous tissue by fibrous connective tissue The fibrous connective tissue which invades the stratum corneum tends to become frayed from friction and appears cauliflower-like (Fig 149) It resembles verrucae, so that when the lesion is mistakenly treated, the scar tissue increases If the erroneous treatment is repeated, as it often is, the lesion becomes intractable

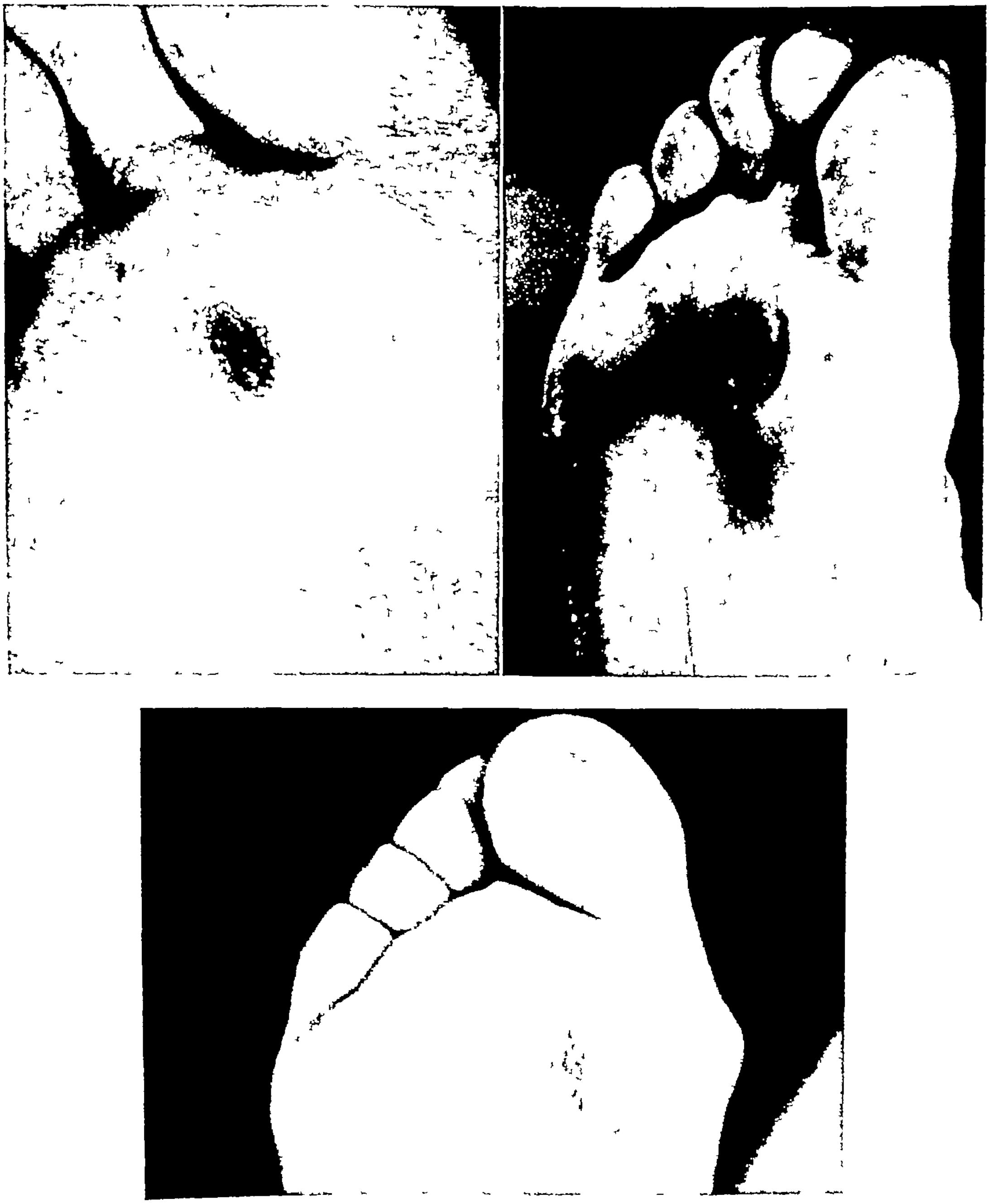


Fig 149 —Different types of plantar keratosis under metatarsal heads, resembling verruca plantaris

imal end of the amputated condyle is often held in place by the fibers of the transverse metatarsal ligament which must be severed before the fragment can be removed. This is best done with small DuVries-modified Sistrunk scissors* inserted into the fragment and the metatarsal head in order to sever the fibers.

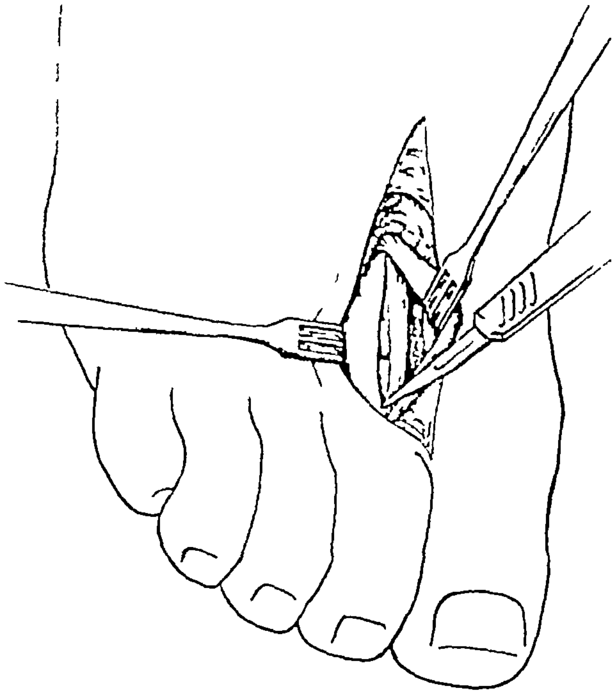


Fig 152—Incision made from middle of metatarsal shaft to web Skin and extensor tendon retracted, capsule incised longitudinally (From DuVries, H L J A M A 152·1202, July 25, 1953)

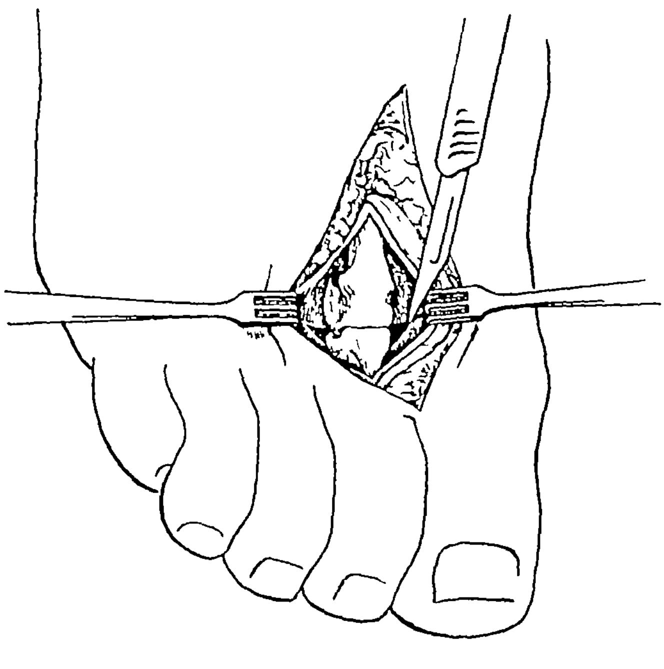


Fig 153—Capsule on both sides of metatarsal head sectioned vertically (From DuVries, H L J A M A 152 1202, July 25, 1953)

*Available on special order at manufacturing companies

quent plantar radiodermatitis resulting after improper diagnosis of plantar lesions. Brown and Fryer (1951) believe that plantar warts comprise most repairs of surface defects of the sole.

Drastic operations have been recommended. Dickson (1948) suggested excision of the entire metatarsal shaft, together with the dorsal and plantar soft structures covering it, including the toe (Fig. 150). Blair (1937) advised excision of the growth and its surrounding skin followed by pedicle grafting from a nonweight-bearing surface of the sole of the foot. Giannestras (1954) shortened the metatarsal shaft by a stepped-down osteotomy (Fig. 151). McKeever (1952) stated that an arthrodesis of the first metatarsophalangeal joint corrects many of the cases.

No method has been found whereby one can visualize accurately plantar condyles on a roentgenogram. From whatever angle the roentgenogram is taken, the plantar condyles either appear distorted or are not seen at all. If the keratosis is located directly under a metatarsal head, it may be assumed that the condylar process is the causative factor. The relationship of the keratosis to the condylar processes can be positively ascertained by comparing two distances on a dorsoplantar roentgenogram of the foot: one from the keratosis to the end of the toe immediately distal to the keratosis, the other from the keratosis to the medial or lateral border of the foot. Another method is by comparing the measurements on a roentgenogram of the foot taken after a metallic substance has been painted on the keratotic area (Fig. 45, *B*).

Recommended Technique for Plantar Condylectomy.—Excision of the plantar condyles because of intractable keratosis under one of the middle three metatarsals, performed over an eight-year period in more than 300 patients, aged 19 to 64 years, warrants recommendation of the procedure. About 75 per cent were women, about 3 per cent had had bilateral involvement. The site in 49 per cent was under the second metatarsal head, in 36 per cent, under the third metatarsal head, and in 16 per cent, under the fourth metatarsal head.

1. Under general anesthesia and hemostasis controlled with a latex bandage or pneumatic cuff, make a longitudinal incision extending from the middle of the shaft of the metatarsal over the involved metatarsophalangeal joint (Fig. 152) to the web on either side of the toe.

2. Retract the skin and extensor tendon, and incise the capsule longitudinally. In cases of severe hammertoe, the extensor tendon must be sectioned, then retract the capsule along with the skin margins and make a vertical incision in each side of the joint capsule (Fig. 153). That frees the head of the metatarsal so that it can be delivered dorsally.

3. The involved toe is plantar-flexed by the thumb of the surgeon's left hand, while the index finger of the same hand is applying pressure on the plantar surface of the metatarsal (Fig. 154). This delivers the head of the metatarsal out of the wound.

4. Amputate the plantar condyle longitudinally with sharp, pointed bone forceps or an osteotome (Fig. 155), and remove (Figs. 156 and 157). The prox-

After the fibers have been severed, scoop out the amputated fragment with the closed tip of the scissors

5 Smooth the surface and round the edges with a Joseph nasal rasp (Fig. 158) Spicules of bone on the cut surface of the metatarsal or chips of bone on the floor of the wound should be removed at this point

6 Replace the metatarsal head within the joint and close the capsule with interrupted catgut sutures Suture the skin margins as usual

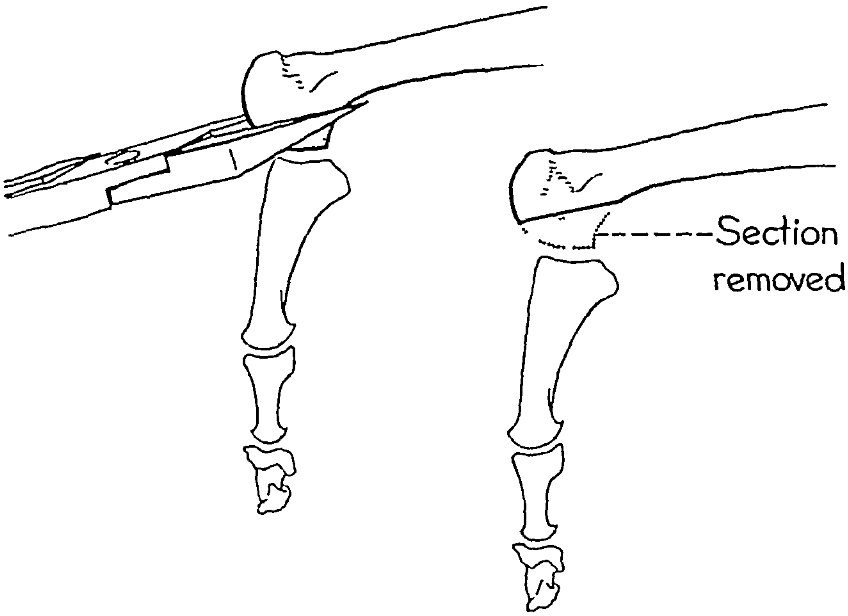


Fig 156 —Section of bone removed

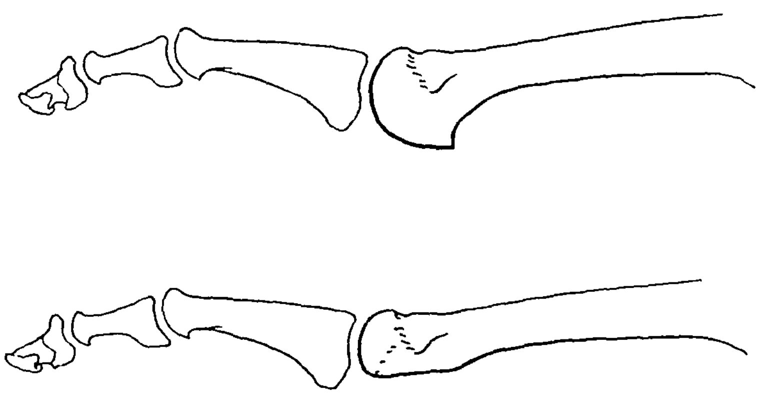


Fig 157 —Before and after removal of plantar condyles

Postoperative Care —Ambulation in a cut-out shoe is permitted on the second postoperative day and is increased daily In about three weeks the patient is able to wear regular shoes to which a Thomas bar has been applied For a few months, residual callus should be removed at regular intervals to keep it from perpetuating itself and to allow healthy tissue to take its place

Results —Follow-up of most cases for six months or longer revealed that about 70 per cent of the patients had obtained complete relief and that keratosis had disappeared in from three to six months There has not been a recurrence

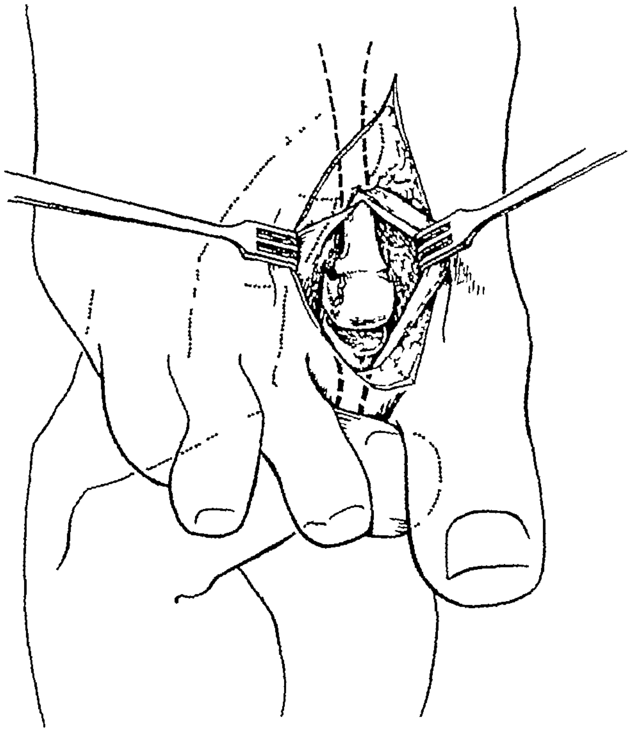


Fig 154.—Toe involved is plantar-flexed by surgeon's thumb of left hand while index finger of same hand applies pressure against metatarsal shaft (From DuVries, H L J A M A 152 1202, July 25, 1953)

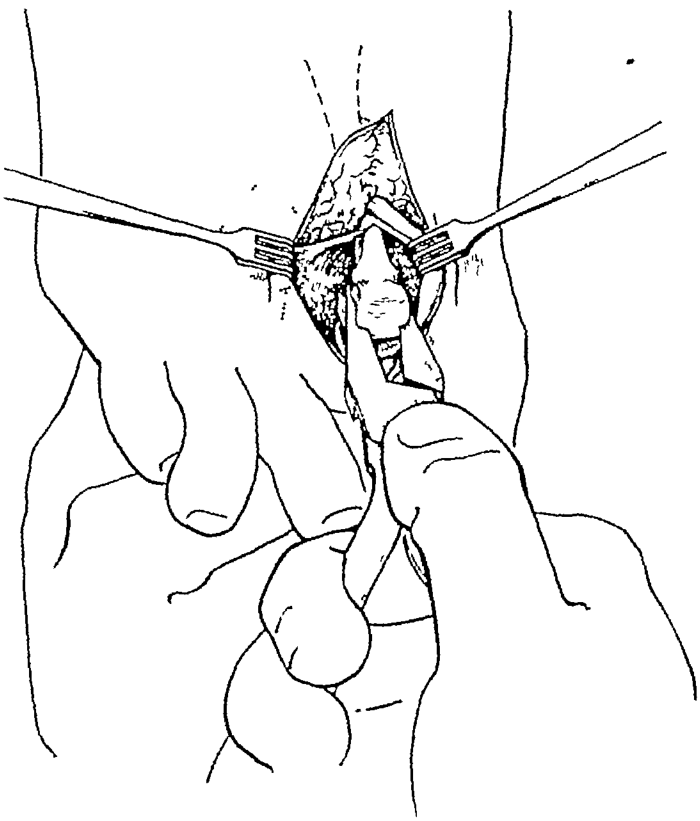


Fig 155.—Plantar surface of metatarsal head excised with sharp-pointed bone forceps or an osteotome (From DuVries, H L J A M A 152 1202, July 25, 1953)

in most cases after as long as ten years About 25 per cent of the patients are improved but have some residual keratosis requiring mechanical aid, such as Thomas bars or inlays, occasional chiropodical treatment is helpful Among the 30 per cent who did not obtain complete relief, about 3 per cent required a secondary excision of the lesion itself, the technique for which is described under Excision of Verrucae Plantaris (page 182) Anderson (1957) performs a rotation flap graft (Fig 159), in addition to plantar condylectomy, in a one-stage procedure This might be advantageous in selected cases Only about 2 per cent of the patients in the series of 300 did not obtain any relief from surgical intervention. All among those 2 per cent had received the maximum amount of irradiation elsewhere preoperatively, from which they had suffered first or second degree roentgen-ray burns Poor results were in direct relation to the amount of previous irradiation

Intractable Keratosis Under First Metatarsal Head.—Although intractable keratosis under the first metatarsal head is common, in general, it is not so disabling as when it occurs under the middle three metatarsals Most instances are under the tibial sesamoid In rare cases, the fibular sesamoid is the offending ossicle The tibial sesamoid normally assumes most of the weight-bearing function transmitted to the head of the first metatarsal Its articulation with the tibial facet under the head of the first metatarsal is unique in that this facet is concave and the dorsal surface of the tibial sesamoid is convex. Because the sesamoids are embedded in the tendon of the flexor hallucis brevis, which inserts into the base of the proximal phalanx, any degree of hallux valgus tends to rotate both sesamoids on the long axis The fibular sesamoid tends to rotate into the first metatarsal interspace, thereby disposing of the possibility of its becoming a weight-bearing focus, whereas the tibial sesamoid may rotate on its side and thus become a weight-bearing pivot The sesamoids may present among their varied shapes unusual thickness or sharp points A sharp ridge or

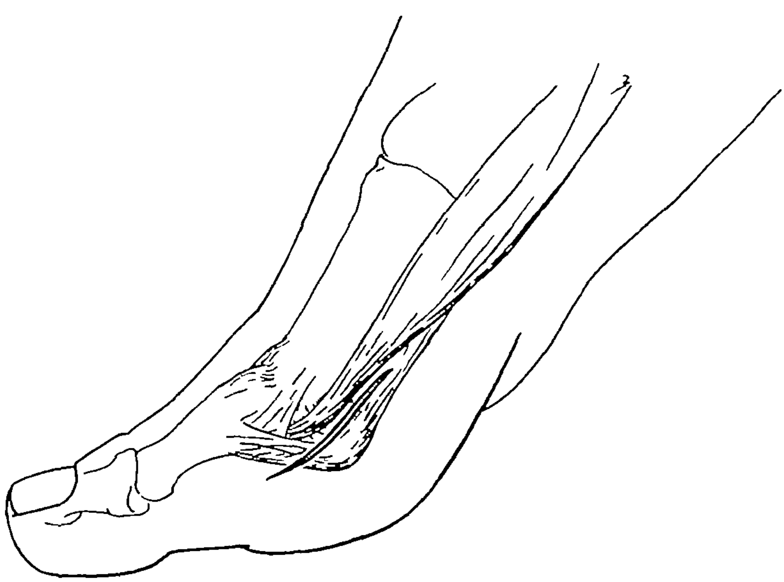


Fig 160—A semielliptical incision follows medioplantar border of first metatarsophalangeal joint

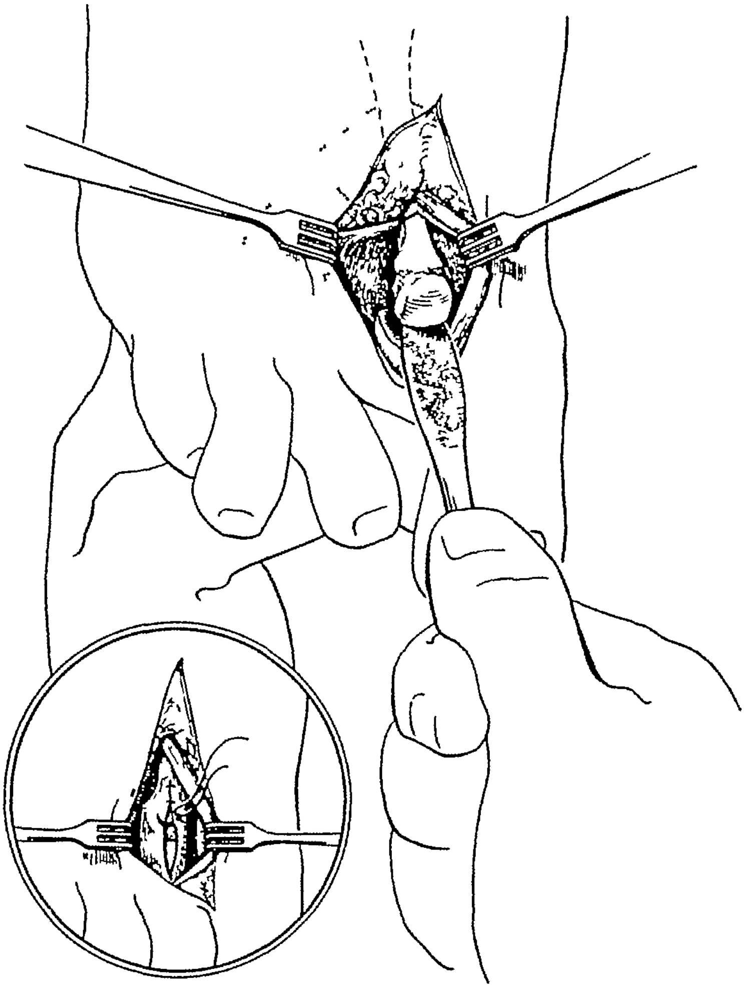
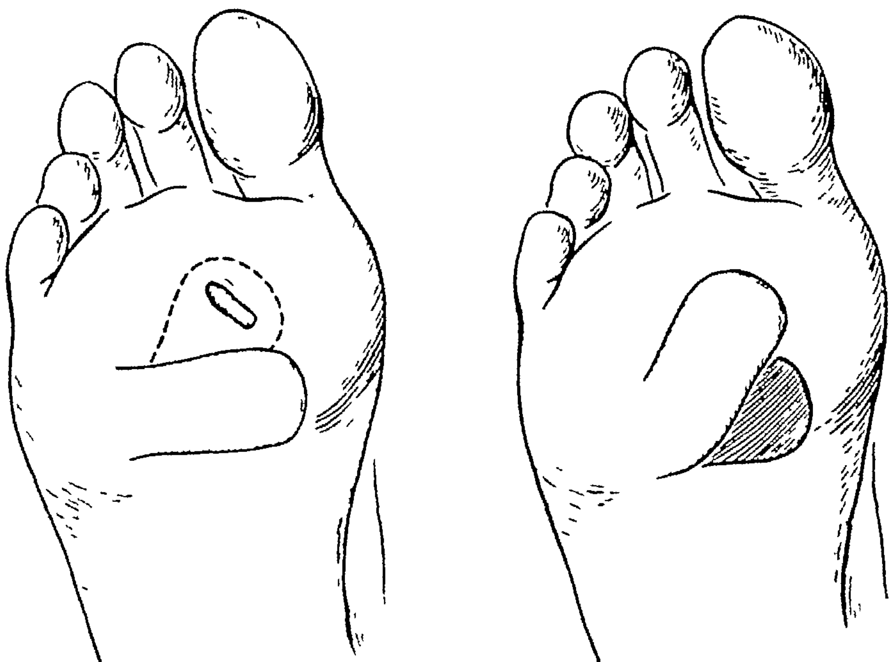


Fig 158 —Surface where bone was removed smoothed with nasal rasp (From DuVries, H L.: J A M A 152 1202, July 25, 1953)

A



B

Fig 159 —Pedicled flap to cover excised area *A*, Keratotic lesion excised Transverse pedicle flap formed immediately below it *B*, Flap graft rotated to cover excised area (After Anderson)

2. Dissect and retract the plantar skin and subcutaneous tissue from the plantar surface of the great toe joint (Fig 160)

3 Place the index finger against the plantar surface of the sesamoid while the great toe is dorsiflexed This reveals the outline of the tibial sesamoid (Fig 161).

4 Make an incision about 1 cm long over the mediosuperior surface of the sesamoid (Fig 162) Grasp the plantar margin of this incision, including the superior margin of the sesamoid, with Allis forceps (Fig 163)

5 Remove the sesamoid by sharp dissection, using its own outline as a guide

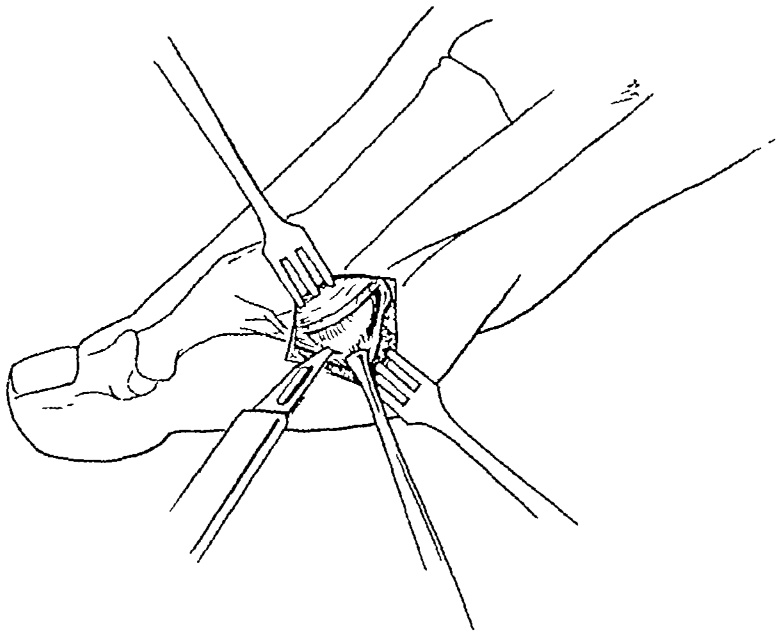


Fig 163 –Periosteum of sesamoid grasped with Allis forceps Sesamoid excised by sharp dissection

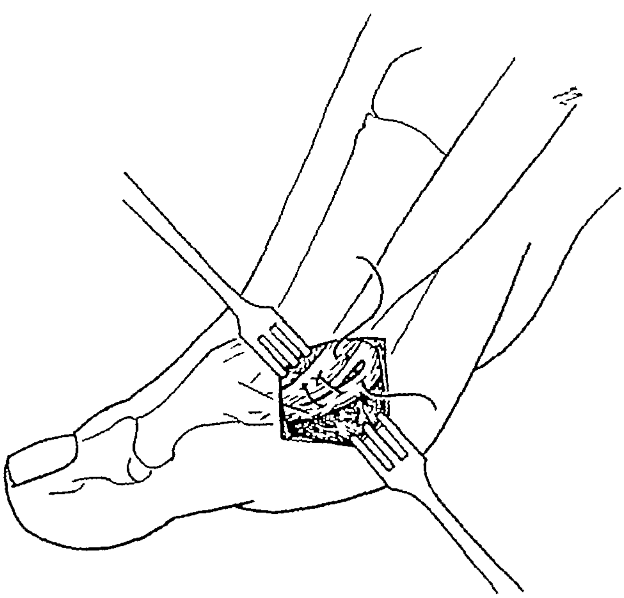


Fig 164 –Fascia closed with interrupted catgut sutures and skin closed in layers as usual

point on its plantar surface may cause a deep-seated callosity to form under it. That callosity is also often erroneously diagnosed and treated as verrucae plantaris and consequently may also become an intractable lesion.

Recommended Technique for Excision of Tibial Sesamoid.—Excision of the offending sesamoid in 300 cases over a span of twenty years gave complete relief in most cases, without postoperative disability in any.

1. Under hemostasis, make a semielliptical incision along the contour of the medioplantar border of the great toe joint, extending the incision from the middle third of the metatarsal to the middle of the proximal phalanx.

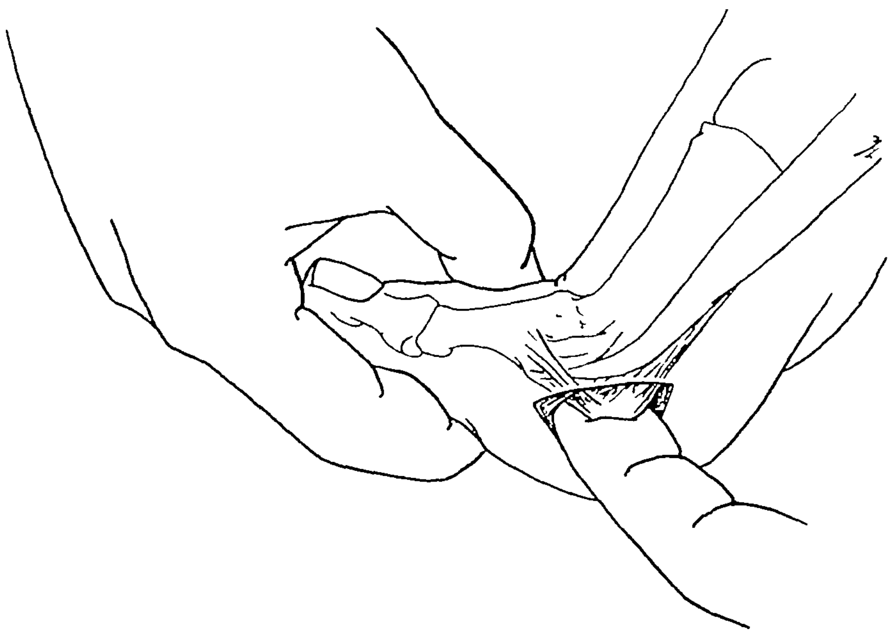


Fig. 161.—Plantar skin margin retracted. Surgeon's right index finger inserted into wound. When great toe is dorsiflexed, sesamoid glides over index finger, pinpointing sesamoid.

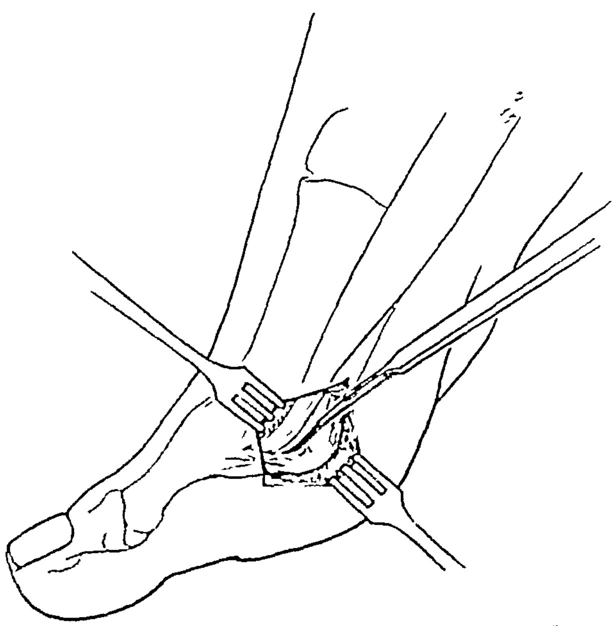


Fig. 162.—Incision into the capsule along mediosuperior surface of sesamoid.

Epidermal cysts are often erroneously diagnosed because they are relatively uncommon and because the skin surface may appear normal A diagnosis is made by finding, on palpation, a freely movable circumscribed subdermal mass of medium hardness

Excision of the mass is simple When possible, the incision should be made on a nonweight-bearing surface If the growth is immediately under a metatarsal head, the incision should be made on either side of the cyst, so that if a scar forms at the incision, it will be under the metatarsal interspace Epidermal cysts are readily shelled out, the thick white glistening capsule is only loosely adherent to the surrounding tissue After the skin incision has been made, the cysts can be extruded by applying pressure on the sides of the mass

Fig 165

Fig 166

Fig 165 —Epidermal cyst on ball of foot which became the most prominent weight-bearing area

Fig 166 —Large epidermal cyst formed on dorsum of distal phalanx after partial amputation of this bone

If a large space is left by the removal of the cyst, it must be closed to prevent formation of a hematoma This is accomplished by suturing the skin subdermally to the fascia with fine catgut before suturing the skin margins Another method is to apply a thick plug of gauze over the sutured incision, where it is held in compression for twenty-four hours Healing is by first intention Ambulation may begin in twenty-four to forty-eight hours

6 Close the capsule and fascia with interrupted catgut sutures, and close the skin as usual (Fig 164).

7 A compression bandage is applied for twenty-four hours Ambulation may begin in forty-eight hours

Technique for Excision of Fibular Sesamoid—The technique for excision of the fibular sesamoid is described under Hallux Valgus, page 383

Keratosis Under Fifth Metatarsal Head.—Keratotic changes under the fifth metatarsal head rarely become intractable because the plantar condylar surface of the head of this bone is usually flat and broad, giving it a good weight-bearing area When a callus does form, it is widespread and is most frequently associated with functional foot disabilities which force the patient to bear excess weight on the outer border of the foot The callus usually disappears after the disability has been corrected In a few instances the condylar process of the fifth metatarsal head becomes distorted or pointed When that happens, the problem is comparable to distortion under the other metatarsal heads The procedure for condylectomy follows

1 Under hemostasis, make a linear incision along the lateroplantar border of the fifth metatarsophalangeal joint

2 Free the plantar skin margin from the plantar surface of the head of the fifth metatarsal

3 Palpate the plantar condylar surface of the bone through the capsule If sharp projections are present, incise the capsule on the lateral side of the joint and retract the plantar margin, thereby exposing the plantar condyle, which is removed with bone forceps or an osteotome, and smooth the cut surface with a rasp to form a flat surface, however, if projections cannot be felt through the capsule, it is safe to close the wound Merely freeing the skin and subcutaneous tissue from the plantar joint capsule often is sufficient to make the plantar excrescence disappear

Epidermal Cyst

An epidermal cyst is an encapsulated mass filled with caseous material; it is often erroneously called *sebaceous cyst* Epidermal cysts appear on any part of the body, oftenest on the scalp, occasionally on the plantar surface (Fig 165) of the foot There are no sebaceous glands in the plantar surface, hence, sebaceous cysts cannot form there On sectioning many so-called sebaceous cysts, glandular epithelium was absent, and the caseous material did not contain fat Invagination and destruction of the epidermis were typical Epidermal cysts on the plantar surface vary in size from 0 5 to 3 0 cm They are painful only when they form on a weight-bearing surface, such as on the ball of the foot, where they do form commonly. They also form on other areas of the foot, however, even on the dorsum of a hammertoe and in old incisions (Fig 166) They are extremely painful under a metatarsal head, as if a pebble were in the sole of the foot The cyst sometimes degenerates and becomes infected, producing a discharging sinus.

itself must be kept clean Rough handling or the application of chemical irritants is likely to worsen the degenerative process Only in extreme cases is surgical intervention indicated and then only by the most conservative procedure applicable

Fibromatosis of the Skin Over Tendo Achillis

Fibromatosis over the tendo achillis is an accumulation of connective tissue of the skin over the tendo achillis, immediately above its insertion The condition often referred to as *tendo achillis bursitis* develops in persons in whom the upper posterior border of the calcaneus juts out acutely posteriorly This phenomenon is sometimes referred to as *Haglund's disease* (page 125) A heel bone thus shaped makes the contour of the back of the heel a straight line

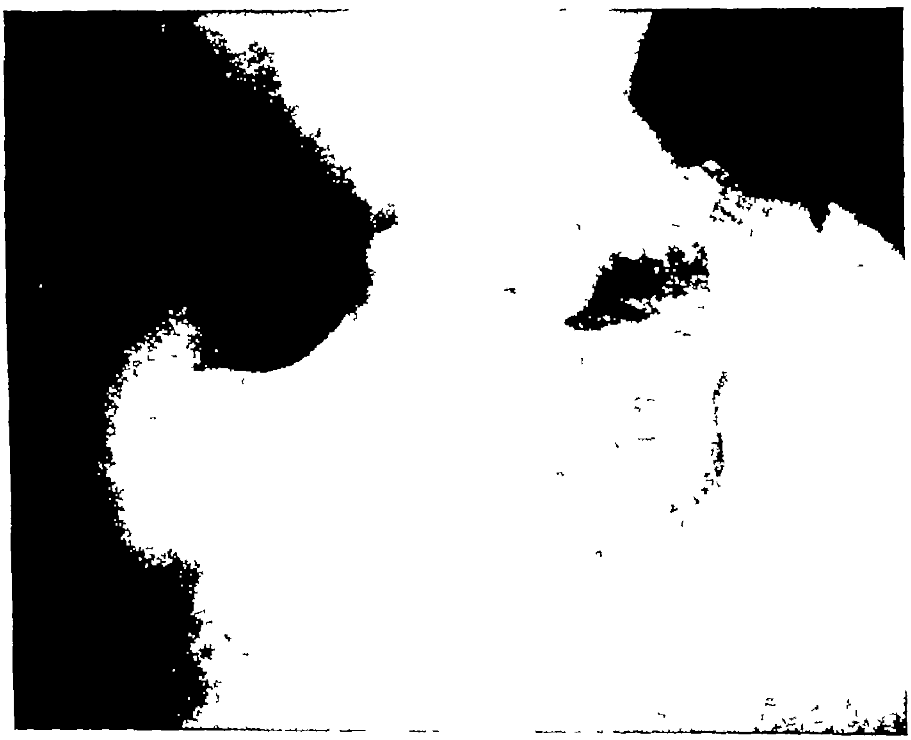


Fig 167 —Fibromatosis of insertion of tendo achillis as result of prominent posterosuperior tuberosity of calcaneus

(Fig 167) The shoe counter is always convex, therefore, its upper margin exerts an abnormal amount of friction and pressure on this prominence The condition is commoner in women as a result of wearing pumps and high heels The growth is solid connective tissue in which a cavity appears only if the tissue breaks down, producing a draining sinus

The growth may be excised through an incision on one side or the other, it does not matter which, of the tendo achillis The mass is not encapsulated and must be dissected with great care, otherwise the skin may be irreparably injured The skin should be sutured subdermally to prevent dead space and consequent formation of hematoma A plaster walking cast is applied with the foot in a mild plantar-flexed position A window in the cast over the field of

Suppurating Sinuses of the Foot

Suppurating sinuses of the foot are newly formed channels lined with granulation tissue, they discharge a seropurulent material The sinus may form in a corn or callus over a bony prominence Such suppurating sinuses ordinarily are chronic, but acute exacerbations are typical

Etiology.—A suppurating sinus of the foot is produced in the same way that corns and calluses are, by persistent sharp pressure over a bony prominence which devitalizes the tissue under the excrescence, and, in some cases, necrosis of the central portion takes place The presence of a debilitating disease worsens the degenerative process

Site—Suppurating sinuses of the foot occur in the following areas under a hard corn on the lesser toes, in the center of a soft corn, generally in the web between the fourth and fifth toes, on the medial side of the great toe joint, usually over the tibial condyle of the first metatarsal head, and often associated with a hallux valgus deformity, especially if there is a sharp projection on the tibial side of the head where they may penetrate into the joint space and cause degeneration of the articular surface immediately under the sinus, in rare instances, digression of the sinus along the shaft of the first metatarsal, on the fibular side of the fifth metatarsal head, associated with tailor's bunion

A degenerative sinus may occur under any of the metatarsal heads, especially the middle three In the young and middle-aged, they are often secondary to unusually large condylar processes on the plantar surface of the head of the metatarsals Under the first metatarsal they are the result of an unusually large or malshaped sesamoid Clawtoe or clawfoot may produce such a sinus under a metatarsal head In older persons such a sinus is often associated with a degenerative disease, such as diabetes Trophic changes due to peripheral nerve disease also produce such a sinus, typically under the head of the first metatarsal They usually retrogress, producing extensive ulceration (See Trophic Ulcers, page 124) Under the head of the second metatarsal a degenerative sinus often results from static dislocation of the second metatarsophalangeal joint (page 404)

Treatment—When the sinus is over a nonweight-bearing area and is not complicated by a degenerative disease, excision of the condyle or bony prominence beneath the sinus without excising the necrotic tissue disposes of the problem in most cases Excision to eliminate the sinus itself may or may not be required

On the plantar surface, in cases such as hammertoe or metatarsophalangeal dislocation when debilitating disease is not a complication, healing usually takes place after open reduction of the deformity underlying the sinus or after excision of the offending plantar condyle or offending sesamoid

Patients in whom there is an underlying degenerative disease should be treated conservatively by such mechanical measures as balanced weight distribution by the use of shoe inlays, Thomas bars, shoe wedging, and padding Total weight bearing should be reduced as much as possible Close cooperation with the internist to improve the general status is important The sinus

for several months and then to administer an erythema dose of roentgen rays A month later, the scar may be excised and from 0 5 to 1 ml 25 mg. hydrocortisone solution instilled into the wound. The skin is underscored for some distance, so that there will be minimal tension on it. If possible, all suturing should be subdermal. Additional irradiation is indicated when there is any threat of recurrence.

Ainhum Disease

Ainhum derives its name from an African dialect, from a word meaning compression. The disease is a diastasis *(dactylolysis spontanea)*, usually of the fifth toe, rarely of the other toes It is characterized by a spontaneous formation of a constricting ring near the proximal phalangeal joint, which strangulates the distal portion of the toe until it falls off. The ring appears first on the plantar surface and finally produces complete encirclement (Fig. 168) This is primarily

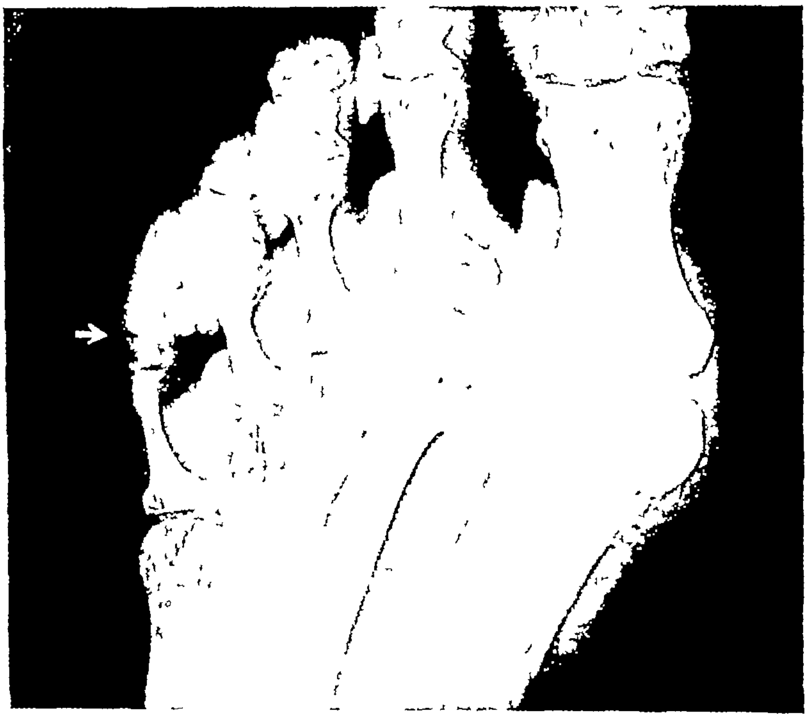


Fig 168 —Ainhum's disease Note constricting band and destruction of middle phalanx

a tropical disease, especially among the Negroes of Africa, but it has been observed in the southern part of the United States (Spinzig, 1939, Stack, 1950), rarely in temperate regions It was first described by Da Silva Lima in 1867 Tye (1946), Norton and his associates (1957), and Aukland and his associates (1957) have contributed thorough reviews of the publications on the subject

The cause is unknown It is commoner in men and is believed to be due to local endarteritis. The disease is localized and without symptoms. The toe drops off in about two years There is no known treatment

Dermatitis Pedis

The vast variety of dermatoses which may involve the foot is not within the scope of this text The common dermatophytosis (Dolce, 1944, Baer and co-work-

operation permits redressing. The cast is removed in about five weeks. Dorsiflexion is begun gradually.

Keloid and Hypertrophic Scar

Keloids.—Keloid is an idiopathic new fibrous mass of the stratum corium raised above the skin surface anywhere on the body. It is typically regular in outline, often appearing multiple, varying in size from a pinhead to massive proportions. Keloids are white or reddish globular masses, either round or oval. A single lesion or many, even dozens, may appear. Keloids form between the ages of 10 and 19 years. They rarely form in older persons. The tendency to keloid formation is not constant throughout life but may come and go. Dark-complexioned persons apparently are more susceptible to keloid formation.

Symptoms.—Keloids become painful only from secondary irritation; they are otherwise symptomless.

Treatment.—For true keloids, Kitlowski (1953) advises an erythema dose of roentgen rays followed by partial excision in about a month, done with strict precaution to ensure primary union, and without suturing the normal skin. Some patients require additional postoperative roentgen therapy. A high percentage of recurrences has been reported after excision alone when the recurrent keloid is larger than the original.

Hypertrophic Scar.—Hypertrophic scar is a false keloid, found only in healing wounds. It is limited to the area of injury and follows the outline of the original injury, but the growth is raised above the surface of the skin. Histologically, hypertrophic scar cannot be differentiated from true keloid. It is symptomless except when the tissue is irritated by friction or pressure. It can become annoying on the foot, especially when it appears on a weight-bearing surface.

Etiology.—The cause of the hyperplasia is not clear. Hypertrophic scar seems to be related to excessive irritation to a wound. Exposure to radiation of an atomic explosion tends to precipitate hyperplasia. Excessive tension in a wound, burns, severe lacerations, strong chemicals, and excessive surgical handling—each induces hyperplasia.

Treatment.—A hypertrophic scar can usually be softened and reduced in size by rubbing castor oil into it for fifteen minutes daily for several months. This procedure may also be used to differentiate the scar from true keloid, because this treatment has no effect on a true keloid.

Hypertrophic scars may be excised in their entirety; however, without roentgen therapy, a high percentage recurs. Gathings (1954) believes that roentgen therapy is the most satisfactory method. Whitehill (1954), in a series of cases of excision of hypertrophic scar, applied a 1 to 2.5 per cent hydrocortisone ointment in the lower half of the wound but none in the upper half as control. There were no recurrences in the treated half of the wound but some recurrences in the untreated half. Asboe-Hansen and his associates (1956) injected hydrocortisone in fifty-six patients with false and true keloids. Their results were better in the false type.

Recommended Procedure.—The procedure recommended here is to soften the scar and reduce its size by rubbing castor oil into it for fifteen minutes daily

Brown, J B (St Louis), and Fryer, M P Repair of Surface Defects of Foot, J A M A 146 628-632, June 16, 1951
Converse, J M Plastic Repair of the Extremities by Non-Tubulated Pedicle Skin Flaps, J Bone & Joint Surg 30A 163-194, Jan, 1948
Dickson, J A Surgical Treatment of Intractable Plantar Warts, J Bone & Joint Surg 30A 757-760, July, 1948
Dolce, F A Shoe Dermatitis Among Soldiers, Mil Surgeon 95 505-507, Dec, 1944
Ducourtioux, M Plantar Warts and Their Treatment, Presse méd 58 1116, Oct 11, 1950
Dunbar, Flanders Emotions and Bodily Changes, ed 3, New York, 1947, Columbia University Press, pp 401-403
Duthie, D A, and McCallum, D I Treatment of Plantar Warts With Elastoplast and Podophyllin, Brit M J 2 216-218, July 28, 1951
DuVries, H L New Approach to Treatment of Intractable Verrucae Plantaris (Plantar Warts), J A M A 152 1202-1203, July 25, 1953
DuVries, H L Verruca Plantaris, J A M A 154 1302, April 10, 1954
Editorial Treatment of Plantar Warts, New England J Med 248 659, April 9, 1953
Galland, W I An Operative Treatment for Corns, J A M A 100 880-881, March 25, 1933
Gathings, J G Treatment of Keloids With Combination of Hyaluronidase and Kutapressin, Am J Surg 88 429-430, Sept, 1954
Gaul, L E, and Underwood, G B Primary Irritants and Sensitizers Used in Fabrication of Footwear, Arch Dermat & Syph 60 649-675, Nov, 1949
Giannestras, N J Clinical Orthopedics No 4, Philadelphia, 1954, J B Lippincott Co, p 225
Haboush, E J, and Martin, R V Painful Interdigital Clavus (Soft Corn), Treatment by Skin Plastic Operation, J Bone & Joint Surg 20 756-757, July, 1947
Ignatoff, W B Verrucae Therapy, J Nat A Chiropod 44 37-46, July, 1954
Kile, R L How Communicable Are Warts? J A M A 162 1222-1224, Nov 24, 1956
Kitlowski, E A Treatment of Keloids and Keloidal Scars, Plast & Reconstruct Surg 12 383-391, Dec, 1953
McElvenny, R T Corns, Their Etiology and Treatment, Am J Surg 50 761-765, Dec, 1940
McKeever, D C Arthrodesis of the First Metatarso-Phalangeal Joint for Hallux Valgus, J Bone & Joint Surg 34A 133, Jan, 1952
Montgomery, A H, and Montgomery, R M Mosaic Wart An Unusual Type of Plantar Wart, New York J Med 37 1978, Dec 1, 1937
Montgomery, A H, and Montgomery, R M Mosaic Type of Plantar Wart, Arch Dermat & Syph 57 397-399, March, 1948
Montgomery, A H, Montgomery, R M, and Montgomery, D C The Problem of Plantar Radiodermatitis, New York J Med 49 1664-1667, July 1, 1949
Montgomery, R M, and Montgomery, A H Common Hyperkeratotic Lesions of the Foot, J A M A 124 756-761, March 18, 1944
Niles, H D Dermatitis Due to Shoe Leather, J A M A 110 363-365, Jan 29, 1938
Norton, M L, Sala, A M, and Silverstein, M E Ainhum (Dactylosis Spontanea), A M A Arch Surg 75 473-478, Sept, 1957
Reeves, R J, and Jackson, M T Roentgen Therapy of Plantar Warts, Am J Roentgenol 76 977-978, Nov, 1956
Rutledge, B A, and Green, A L Surgical Treatment of Plantar Corns, U S Armed Forces M J 8 219-221, Feb, 1957
Spinzig, E W Ainhum Its Occurrence in the United States With a Report of Three Cases, Am J Roentgenol 42 246, Aug, 1939
Stack, J K Ainhum, J Bone & Joint Surg 32A 444, April, 1950
Tye, Mauray Ainhum, New England J Med 234 152-154, Jan 31, 1946
Verrucae Issue, J Nat A Chiropod 44 17-65, July, 1954
Whitehill, J L Prophylaxis of Surgical Keloids, Arizona Med 11 399, Nov, 1954

ers, 1956) and dermatitis due to irritants from a shoe (Niles, 1938, Gaul and Underwood, 1949) do bear description and are considered in Chapter 4, Infections of the Foot

SKIN GRAFTING

Defects in the skin of the foot, especially on the plantar surface, may become a major problem because the skin and subcutaneous tissue of the sole of the foot form a highly specialized structure otherwise found only in the palm of the hands Once the structure is lost, it cannot be replaced but only substituted The covering of the tendo achillis is also specialized On the dorsum of the foot the skin is similar to other parts of the body

Transfer of skin from the opposite sole might seem desirable, but, as pointed out by Brown and Cannon (1944), in most instances it results only in adding disability to the donor foot Skin transferred from other parts of the body to the sole of the foot does not undergo thickening and keratinization to assume the normal character of the sole but retains its original properties, for example, a transplant from a hair-growing surface will continue to grow hair Because the foot is subjected to almost constant friction and pressure, all skin grafts, especially on the sole of the foot and over the tendo achillis, are in constant danger of breaking down, even though the graft is successful Nevertheless, skin grafting must usually be resorted to when a nonhealing suppurating defect occurs, as from a nonhealing pressure ulcer or a burn from irradiation Skin replacement should be undertaken only by a surgeon qualified to perform plastic surgery

Free thick-split grafts are adequate for small defects, whereas for larger defects, cross-leg flaps, as described by Brown and Fryer (1951), Converse (1948), and Braithwaite and Moore (1949), are preferable

Failure of a graft to heal is usually due to (1) hemorrhage under the flap, most likely from oozing of venous blood, (2) infection of the flap, or (3) necrosis of the flap

REFERENCES

Allington, H V Review of Psychotherapy of Warts, A M A Arch Dermat & Syph **66** 316-326, Sept, 1952

Anderson, R Treatment of Intractable Plantar Warts, Plast & Reconstr Surg **19** 384-388, May, 1957

Asboe-Hansen, G, Brodthagen, H, and Zachariae, L Treatment of Keloids With Topical Injections of Hydrocortisone Acetate, A M A Arch Dermat **73** 162-165, Feb, 1956

Aukland, G, Ball, J, and Griffiths, D LL Ainhum, J Bone & Joint Surg **39B** 513-519, Aug, 1957

Baer, R L, Rosenthal, S A, Litt, J Z, and Rogachefsky, Hymen Experimental Investigation of Mechanism Producing Acute Dermatophytosis of Feet, J A M A **160** 184-190, Jan 21, 1956

Billig, H E, Jr Condylectomy for Metatarsalgia Indications and Results, J Internat Coll Surg **25** 220-238, Feb, 1956

Blair, V P, Brown, J B, and Byars, L T Plantar Warts, Flaps, and Grafts, J A M A **108** 24-27, Jan 2, 1937

Blank, H Treatment of Plantar Warts, Arch Dermat & Syph **56** 459-461, Oct, 1947

Braithwaite, F, and Moore, F T Skin Grafting by Cross-Leg Flaps, J Bone & Joint Surg **31B** 228-235, May, 1949

Brown, J B (St Louis), and Cannon, B Repair of Surface Defects of Foot, Ann Surg **120** 417-430, 1944

onychia in multiple nails. Onychia affects the great toe most frequently, where it is due to trauma from pressure of the shoe and from superimposed secondary infection

Treatment is by relief of all pressure and daily application of a germicide and fungicide dressing In rare instances, the condition may become acute, requiring antibiotic therapy.

PARONYCHIA

Paronychia is an inflammation of the nail groove commonly affecting the great toe but sometimes the lesser toes, at times it extends to the matrix *(onychia)* Unlike onychia, however, paronychia is by itself rarely due to constitutional or skin diseases The condition may vary from mild cellulitis to a state of suppuration after mechanically pressured crowding of the tissue on the side of the nail, such as pressure from the side of the boxing of the shoe. Granulation tissue and extensive ulceration in the groove are characteristic Paronychia is either accompanied by or is the forerunner of so-called ingrown nail

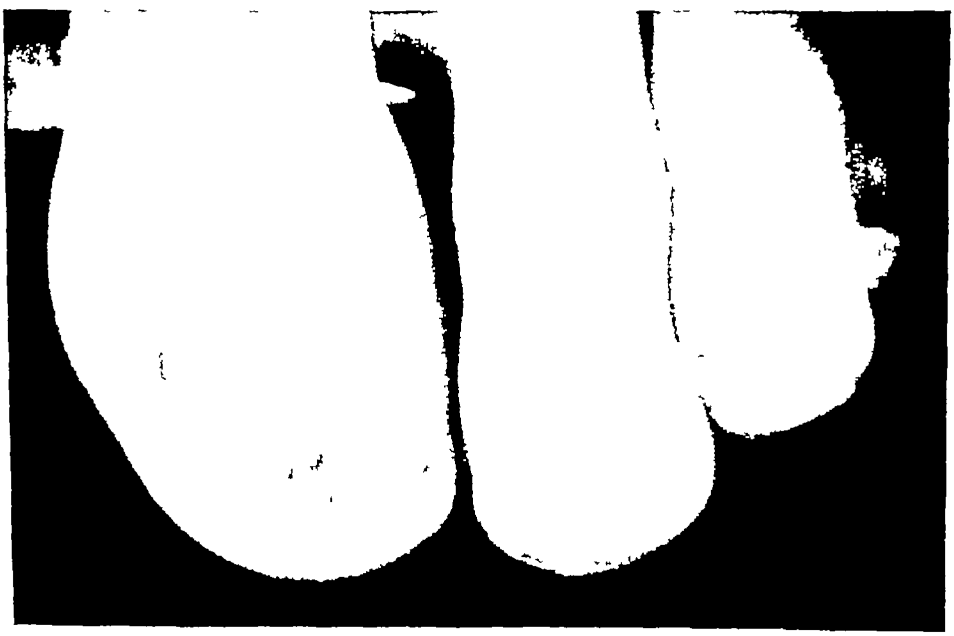


Fig 169 —Congenital double nail of great toe

Treatment consists of the following steps (1) relieve all pressure from shoes, (2) excise a linear portion, about 2 mm of the nail margin, to relieve the cutting effect on the edematous nail groove, (3) paint the granulation tissue with 10 per cent silver nitrate or 90 per cent phenol, (4) apply a fungicide dye and cover with a sterile dressing

ONYCHAUXIS

The hypertrophied nail, termed *onychauxis* or, popularly, *clubnail,* for the most part involves the great toenails, but the nails of the lesser toes may be affected (Fig 170) The deformity may be due to constitutional conditions, such

CHAPTER NINE

Diseases and Deformities of the Toenails

DISEASES OF THE NAIL MAY BE LOCAL OR CONSTITUTIONAL INVOLVEMENTS OR THE result of congenital malformations The nails are cutaneous appendages That is why diseases of the skin and constitutional diseases affecting the skin often also involve the nails White (1939) classifies diseases of the nail as those due to (1) infections, (2) psoriasis, (3) contacts, (4) eczema (atopic dermatitis), (5) hypovitaminosis, (6) tumors, (7) traumatisms, or (8) general diseases Pardo-Castello's (1947) classification covers the divisions according to (1) affections peculiar to the nails, (2) onychodystrophies, (3) ungual manifestations of dermatoses and of systemic disease, and (4) congenital affections of the nails

Congenital nail deviations are errors of development producing such anomalies of the nail as *pachyonychia,* or hypertrophy of the nail and the nail bed, *polyonychia* (Fig 169), and *anonychia* Tauber and his associates (1936) reported a case and reviewed the published reports regarding *pachyonychia congenita,* a rare disease involving the nails, which is accompanied by palmar and plantar keratoses, among other symptoms Krausz (1950), in a review of 2,788 cases of disorders of the nail, reported 94 per cent to be due to local factors, such as trauma or tinea infections

Constitutional diseases involving the nails are in the province of the internist, skin diseases involving the nails are in the domain of the dermatologist Only local and surgical nail problems are considered here For convenience, a glossary of terms pertaining to pathology of the nails is appended to this chapter.

ONYCHIA

Onychia is an inflammation of the matrix, usually extending into the nail grooves *(paronychia)* If the disturbance involves multiple digits, it is ordinarily due to systemic disease In some instances, local tinea infection may cause

dition becomes asymptomatic Treatment as outlined under onychauxis is indicated when there are pain and recurrent infection

ONYCHOMA: NEOPLASMS OF AND AROUND NAIL

Common tumors about the nail are verrucae, periungual fibroma, and subungual exostosis Verrucae and periungual fibromas usually appear in the nail groove but may occur in the nail bed under the nail They respond readily to fulguration or excision Subungual exostosis is discussed on page 152

Malignancy of the nail bed has been encountered and its possibility should not be overlooked Ashby (1956) found reports of twenty-five cases of primary carcinoma of the nail bed and added two proved cases of his own Subungual melanoma, or melanotic whitlow, is a malignancy in the aged often unrecognized

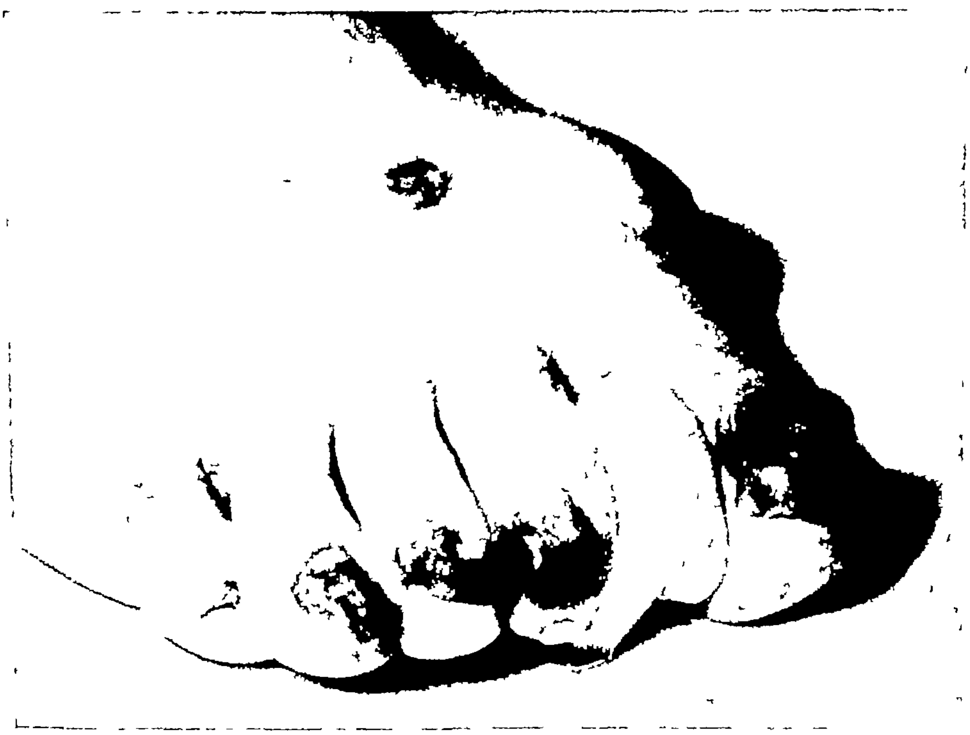


Fig 171 —Ram's horn nails of first four toes

(Gibson and co-workers, 1957), notwithstanding signs which should arouse diagnostic interest increasing pigmentation, chronic nonhealing, painless granular excrescences, persistent splitting of the nail Differential diagnosis is essential Treatment is by local excision

ONYCHOCRYPTOSIS

The terms *ingrown nail* and *onychocryptosis* are misleading because they imply that the side of the nail grows down into the nail groove The unfortunate terminology has given rise to misinformation regarding this common disability Patients are subjected to a series of surgical procedures, only to have recurrences and consequent extensive iatrogenic deformity

as nutritional deficiencies or psoriasis, but most cases are the result of local conditions trauma to the matrix and nail bed or tinea infection, or both trauma and tinea infection, indeed, tinea infection is the most frequent cause

The affected nail and its bed are thickened and deformed When the disorder is due to *tinea gypsum,* the surface of the nail may have white dead streaks or patches, which can be readily excised When the undersurface of the nail contains a yellowish or brown powdery substance, destruction of the nail bed has been by *tinea purpureum*

In about 10 per cent of cases of clubnail, the dorsal distal surface of the distal phalanx undergoes hypertrophic bone changes

Two methods of treatment are available In the first, the hypertrophied nail and bed are gradually reduced by the application of escharotic dressings In the second, avulsion of the nail and repair of the beds are advised The second method is reserved for severely deformed clubnails The method of repair of the bed is outlined under Incurvated Nail, pages 208, 218 to 219

Fig 170 —Ram's horn nail of second toe

ONYCHOGRYPOSIS

Onychogryposis is uncommon It is known also as *ram's horn nail* and *hostler's toe* because of its occurrence in men who tended horses The great toe alone or several toes may be affected (Fig 171) The massive growth folds over the anterior surface of the toe, often terminating on the plantar surface The condition does not appear to have any relationship to tinea infection In most cases, it is due to a congenital tendency and repeated trauma to the matrix associated with poor hygiene, for which reason it is more likely to be seen in derelicts than in those who are careful of cleanliness

The entire horn up to the nail bed and to the matrix is readily removable with a pair of strong nail nippers By frequent subsequent trimming, the con-

ties, the symptoms of which are grouped and called *ingrown nail.* The commonest type is an incurvation of the nail margins

Hypertrophy of the ungualabia (the term preferred over *ingrown nail*), comprises the larger group Normally the space between the nail margin and the nail groove is about 1 mm. The groove is lined with a thin layer of epithelium and lies immediately under and on the sides of the nail margin (Fig. 172). This space is sufficient to protect from irritation under normal conditions. The boxing of shoes, however, often exerts a downward pressure upon the nail plate or upon the nail lip on the side of the toe, and that pressure obliterates the space between the nail margin and nail groove, thus producing constant irritation to the groove The reactive swelling in the groove creates a cycle that results in gradual hyperplasia of the groove and nail lip and ultimate permanent hypertrophy (Fig. 173).

Fig 173 –Hypertrophy and overlapping of nail lip

As the process continues, the nail groove is finally incised by the nail margin, often succeeded by suppuration and secondary infection To relieve the acute symptoms, a triangular section of the nail margin is excised, however, the area left by excision is the forerunner of the *fishhook formation* (Fig 174), about which Frost (1950) speaks Hypertrophy of the groove fills the space of the excised nail margin As the nail continues its growth distally, it collides with the elevated nail groove, resulting in recurrent episodes of infection and massive formation of granulation tissue

Congenitally thick nail lips predispose to *ingrown nail,* which explains why the condition is sometimes seen in infants, or even in the newborn who have

All evidence is to the effect that the growth of the nails in all vertebrates depends on the matrix and that the width of the nail is in direct relation to the width of the matrix (Pardo-Castello, 1947, Maximow and Bloom, 1947) There is no evidence that the matrix becomes wider in persons suffering from this malady The old term *ingrown nail* was chosen on the assumption that ingrowth of the nail, or growing down of the nail into the nail groove, was caused by growth of the width of the convexity of the nail, therefore, early surgeons aimed at destroying the growth of the nail margin, thus, their attempts at correction failed (DuVries, 1933, 1944) The coined term *hypertrophy of the ungualabia* (DuVries, 1933, 1944) is more descriptive of the true pathologic condition Bartlett (1937) also concluded that the terms *ingrown nail* and *ingrowing nails* are misnomers

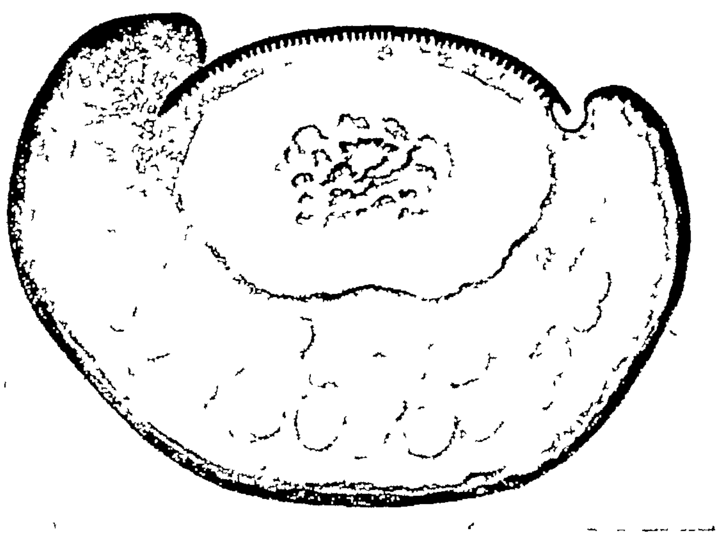


Fig 172 —Left Hypertrophy of nail lip and occlusion of nail groove Right Normal relation of nail margin to nail groove

Frost (1950) recognized three types of so-called ingrown nails (1) a normal nail plate, but as a result of improper nail trimming, a fish hook or spur remains in the nail groove, (2) an inward distortion of one or both lateral margins of the nail plate (*incurvated* nail), (3) a normal nail plate, but the lip is hypertrophied

There are two distinct conditions that produce the symptoms The first is the larger group and accounts for about 75 per cent of the cases It is primarily hyperplasia of the nail groove and nail lip *(hypertrophy of the ungualabia)* The smaller group of 25 per cent comprises essentially a deformity of the nail plate itself It is caused by a malformation of the dorsum of the distal phalanx or by hypertrophy and irregular thickening of the nail bed, often from *tinea unguium* The normal nail plate and its bed is 2 or 3 mm thick, its contour is determined largely by the shape of the dorsum of the distal phalanx The shape and contour of the dorsum of this bone may vary widely because of secondary changes in the bone from irritation and pressure Such variations often produce nail deformi-

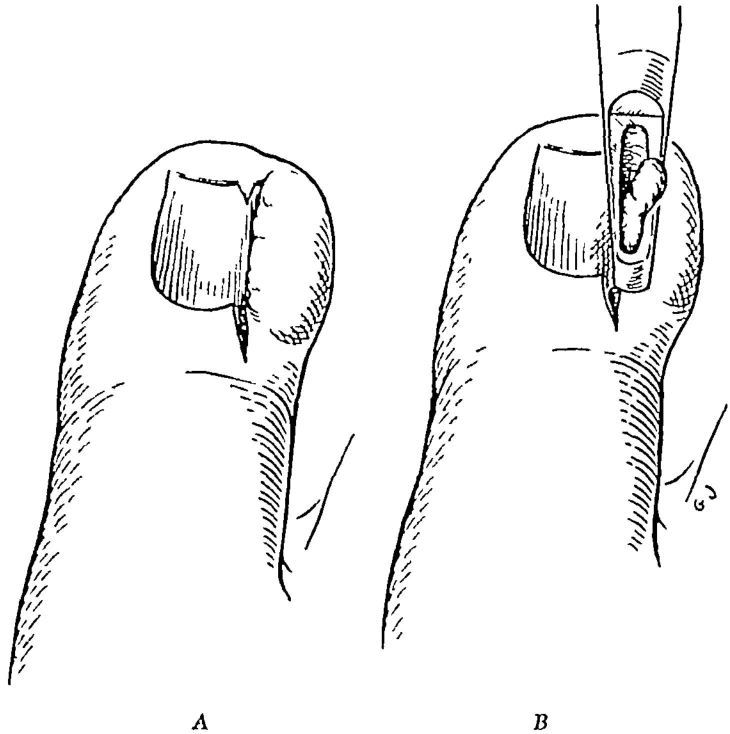


Fig 177 —*A*, Incision over nail matrix *B*, With Steinberg's trephine, nail margin, groove, and part of nail lip removed

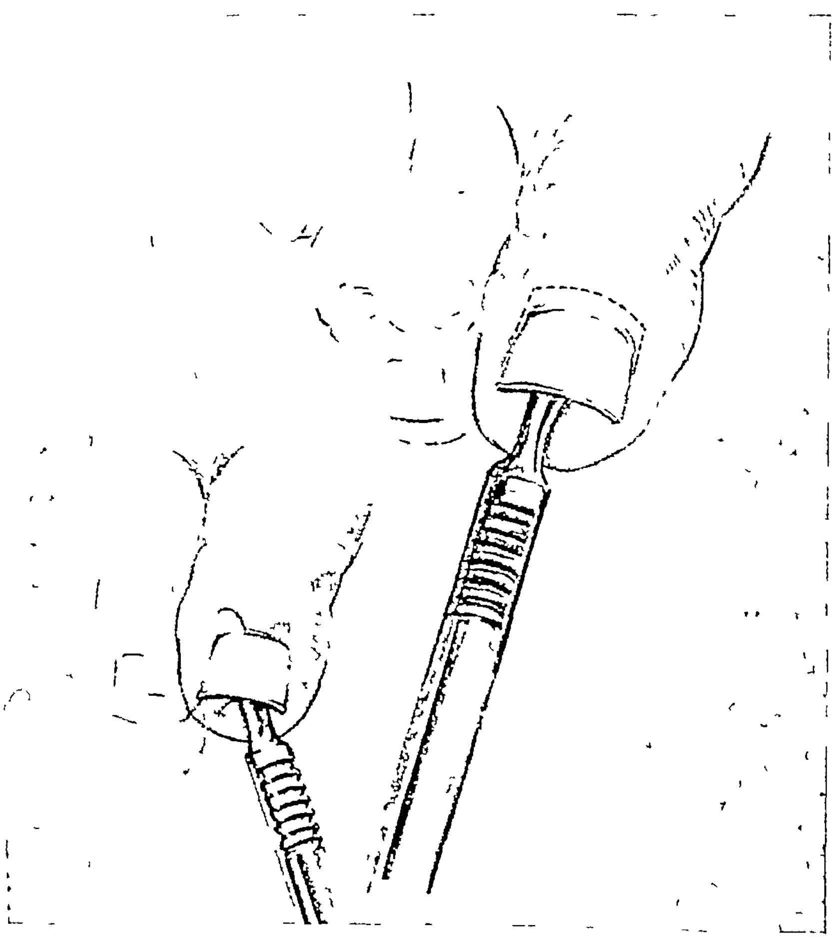


Fig 178 —Nail plate underscored with Frost elevator and nail avulsed

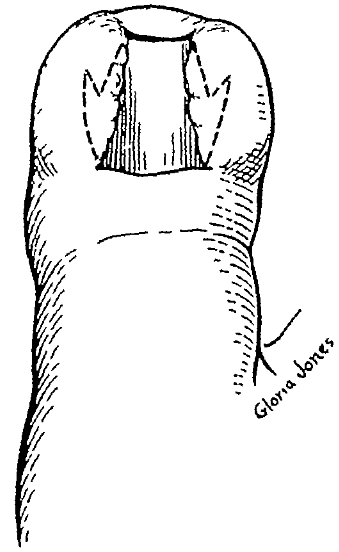


Fig 174 —Consequence of repeated cutting of distal end of nail margin Note fishhook

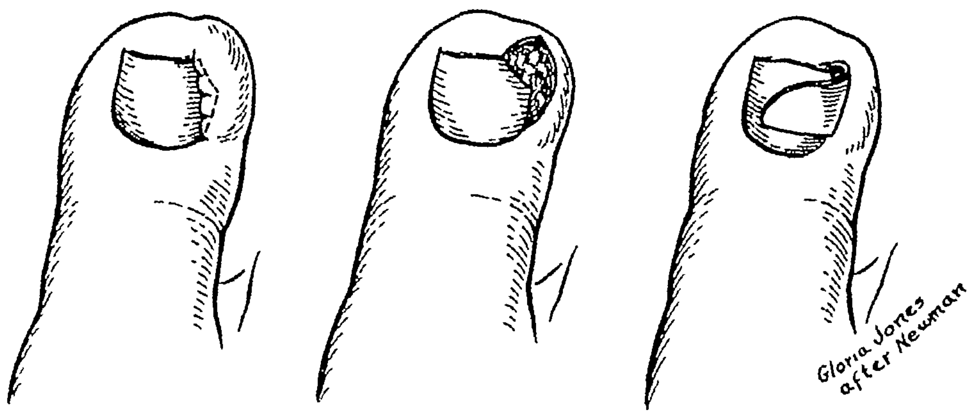


Fig 175 —Newman's appliance for ingrown nail One of many appliances devised for ingrown nail, none of which has proved of value

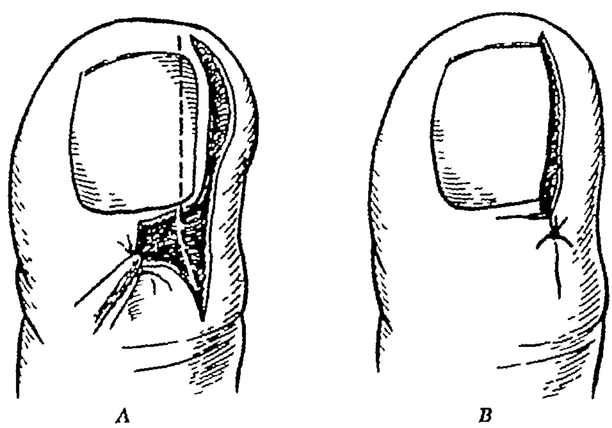


Fig 176 —*A*, Nail margin, groove, and part of nail lip excised *B*, Eponychium sutured over matrix

Recommended Procedure for Hypertrophied Ungualabia.—The treatment for the different stages of hypertrophied ungualabia is as follows

Mild Cases—Relief may be obtained by inserting a few loose strands of cotton in the nail groove so that the nail margin rests upon the cotton to which is applied an astringent, such as 10 per cent silver nitrate Permit the nail to grow out freely over the distal end of the nail groove, this calls for footgear that does not exert constant pressure against the great toe. In the presence of acute cellulitis, with or without infection, the offending nail margin must be excised, a loose cotton packing inserted into the nail groove, and an astringent applied to the cotton pack. This is repeated at frequent intervals until the nail margin becomes completely free up to the distal end of the toe

Moderately Severe Cases—Plastic reduction of the nail lip usually corrects moderately severe cases of hypertrophied nail lip·

1 Remove a spindle-shaped section, about 3 by 1 cm from the side of the nail lip, which is triangular on cross section (Fig 180) The incisions extend from the most distal portion of the toe to about 0 5 cm. proximal to the nail fold, meeting about 2 mm under the nail groove

2 Excise the excess subdermal fat

3 Coaptate and suture the skin margins, drawing the nail groove downward (Fig 181)

The wound heals by first intention, and the procedure results in a free space between the nail groove and margin

Advanced Hypertrophy of the Nail Lip—Advanced cases are likely to be accompanied by large nail lips with excessive granulation tissue in the nail groove. The nail contour is normal, but there is a history of repeated infection of the involved nail groove In a personal series of 300 cases in which the ages of the patients ranged from 6 months to 67 years, DuVries' operation for reduction of hypertrophy of the ungualabia gave uniformly good results

1. Make an elliptical incision, extending from 0 5 cm. proximally to the eponychium to the distal portion of the toe from above downward alongside the nail margin and on the side of the distal phalanx with its base plantarwise The two incisions of the ellipse meet at the base All the hypertrophied tissue of the nail groove and nail lip is thus excised and a skin flap is formed of the side of the nail lip (Figs 182 and 183)

2 Excise all excess fat (Fig 184)

3 Underscore the nail margin for the distance of about 2 mm from its bed all the way to the matrix (Fig 185)

4 Pass a suture through the dorsum of the nail and carry it under the freed portion of the nail and through the deepest portion of the wound, letting it escape through the flap.

5 Return the suture and pass it through the nail from its undersurface (Fig 186).

6 When that suture has been tied, the skin flap is folded under the nail margin (Fig 187)

7 Additional sutures are sometimes necessary at the eponychium and at the distal end of the incision (Fig 188)

thick nail lips or congenital absence of freedom between the nail groove and nail margin, or even both conditions In adults, it is an acquired condition

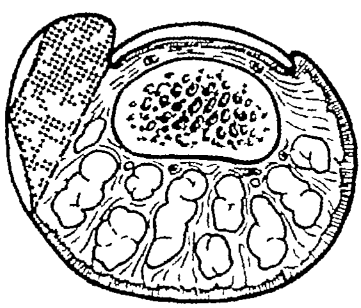
The size, shape, and contour of the nail plate and bed are usually normal Hyperplastic changes of the nail groove and lip are accompanied by formation of granulation tissue on the lip and groove The granulation tissue bleeds freely on slight provocation Hypertrophy may mask a large part of the nail or even most of the nail

The variety of procedures reported for the cure of so-called ingrown nail indicates that none is completely satisfactory, indeed, postoperative recurrence is frequent Keyes (1934) reviewed 110 cases in which five different procedures resulted in fifteen recurrences Unsuccessful appliances have been devised, for example, one by Linch in 1939 and one by Newman in 1949 (Fig 175).

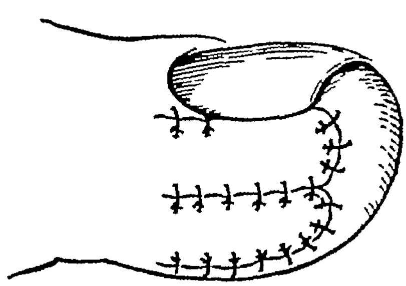
The procedures generally practiced fall into three distinct groups. (1) excision of the nail margin up to the matrix and the nail groove, as illustrated by Winograd (1929), Graham (1929), Jansey (1955) among others (Fig 176), and Steinberg (1954) who devised a punch to simplify the procedure (Fig 177), (2) avulsion of the entire nail, Frost (1958) devised an instrument (Fig 178) to avulse a nail atraumatically, (3) reduction of the hypertrophied lip, as recommended by Bartlett (1937), Ney (1923), and DuVries (1944) The first and second procedures, and their varied modifications, notwithstanding their mutilating and often poor results, are more generally employed than the third procedure, probably because they are simpler to perform than plastic repair of the nail lip, just as digits are often needlessly amputated because it is easier than to repair them.

CORRECTION OF HYPERTROPHIED NAIL LIP

Ney's Operation.—An ingenious operation based on the flap skin graft used in plastic surgery is Ney's procedure Ney forms two skin flaps of the entire surface on the affected side of the toe and then removes all the subcutaneous tissue perpendicular to the nail margin, he replaces the flaps and sutures them to place (Fig 179). The procedure is complicated, and the possibility that flaps will slough off must be kept in mind



A



B

Fig 179.—*A*, Hypertrophied lip and subdermal fat excised at side of toe *B*, Skin on side of toe made into two flaps and sutured

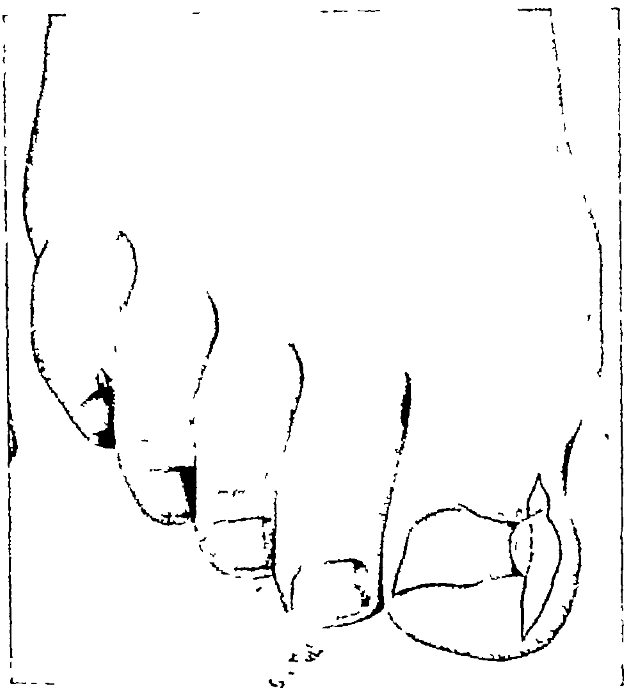


Fig 182

Fig 183

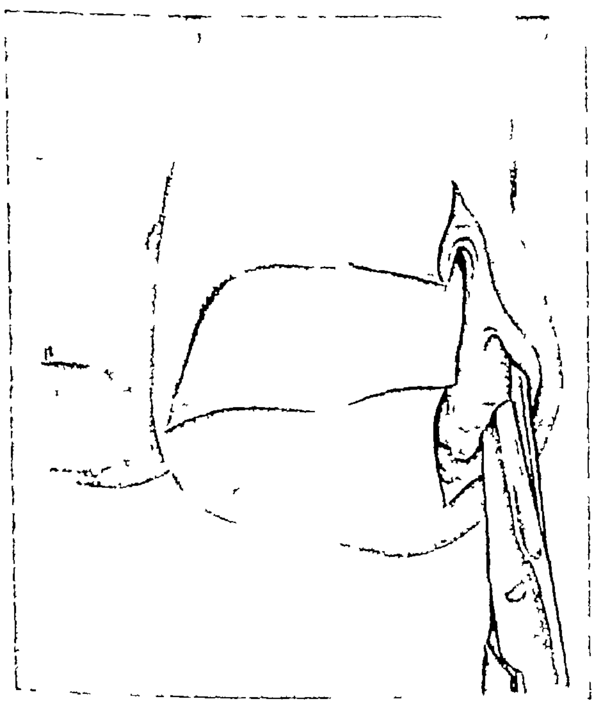


Fig 184

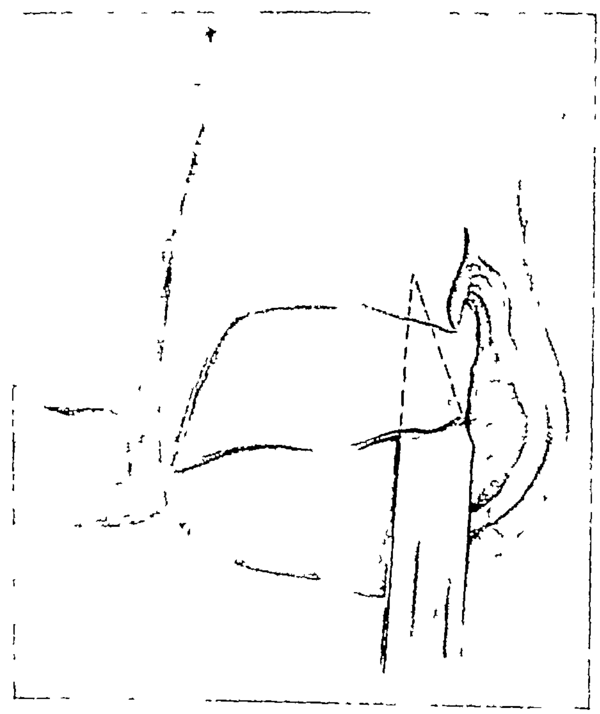


Fig 185

Figs 182 to 189 —DuVries' plastic reduction of hypertrophy of ungualabia

Fig 182 —Linear incision along nail margin A second semielliptical incision made lateral to first incision These incisions to meet at both ends

Fig 183 —Hypertrophied lip removed

Fig 184 —All excess fat excised

Fig 185 —Nail margin detached from nail bed for about 3 mm

The nail margin grows freely over the new groove Fig 189 shows end results

Postoperative Care —Change the dressing in twenty-four to forty-eight hours. If any of the sutures are strangulating, which they often are because of edema, they should be cut but not removed until six to eight days later Thereafter, change the dressing every five to seven days until healing is complete During that period, granulation tissue may form in the wound, if it does, it should be

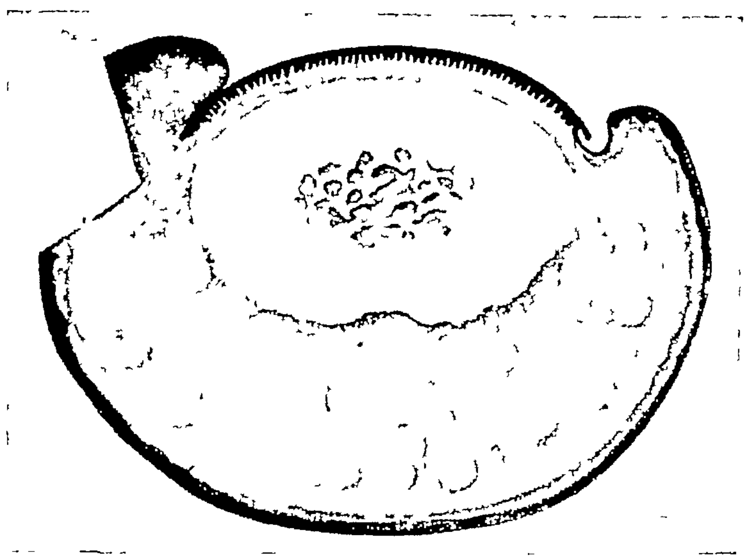


Fig 180 —Triangular section removed from side of toe

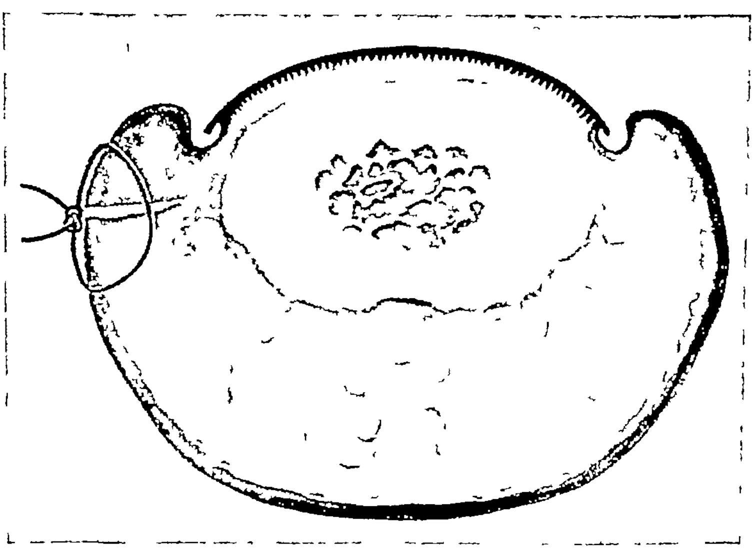


Fig 181 —Suturing of skin margins pulls down nail lip and nail groove

promptly destroyed by painting with 95 per cent phenol or 20 per cent silver nitrate It may be necessary to repeat for a number of visits until healing is assured

The underscored nail margin will not adhere again but will remain detached from the bed, however, the new nail will grow normally because the matrix is

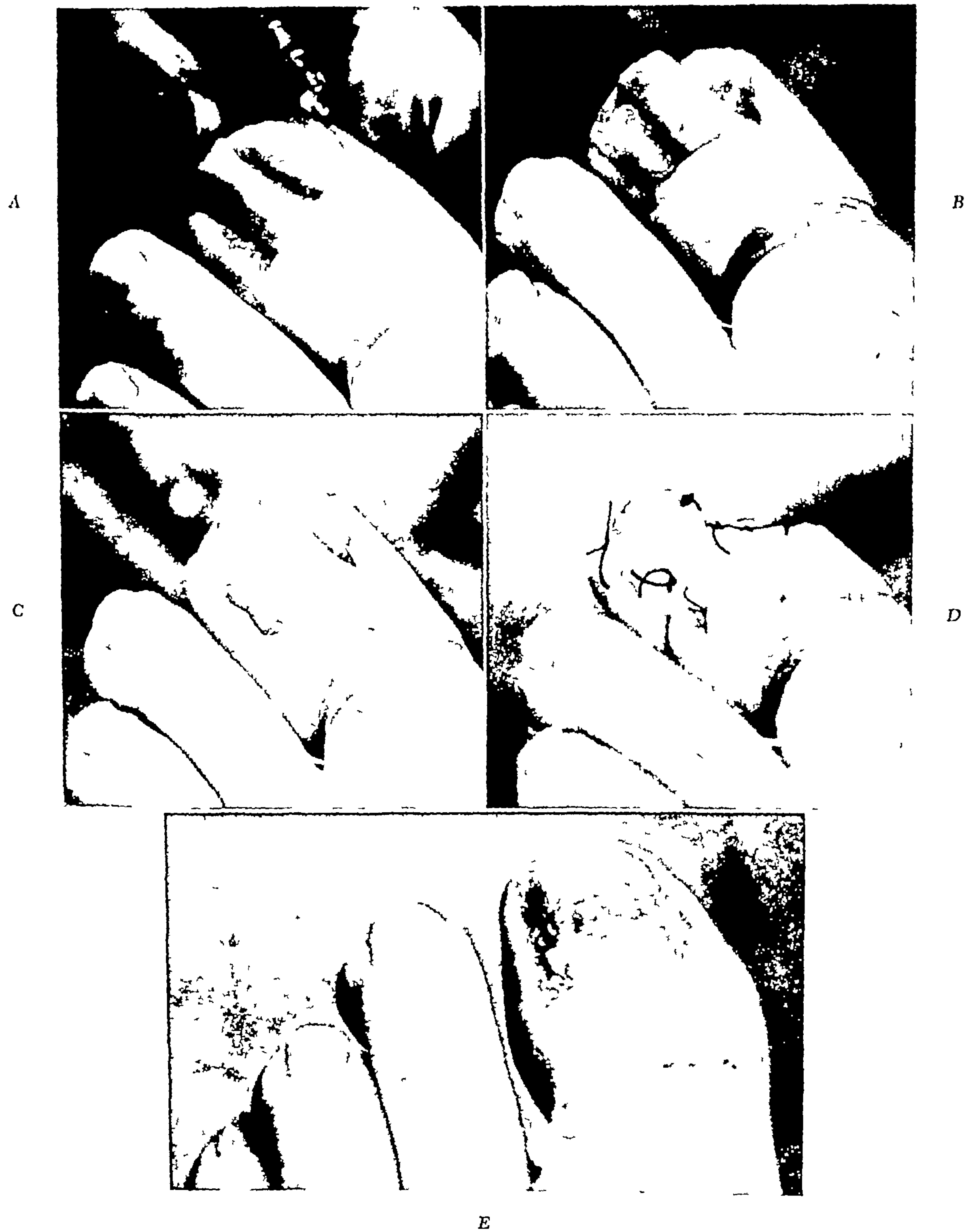


Fig 189—Steps, *A-D*, and end result, *E*, in case in which procedure shown in Figs 176 through 188 was performed

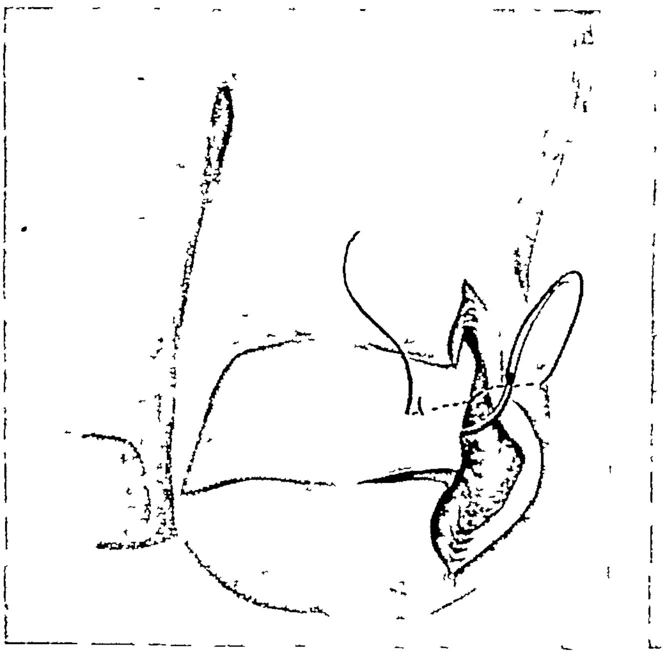


Fig 186

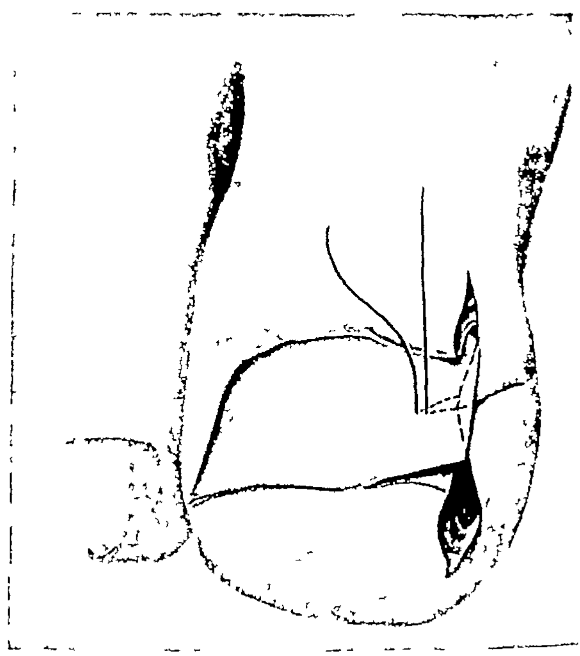


Fig 187

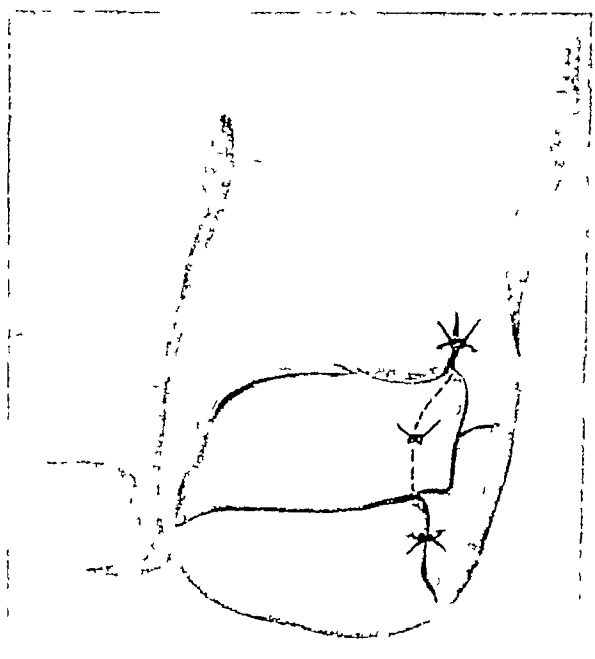


Fig 188

Fig 186 —Suture carried through dorsum of nail plate, under freed nail margin, through floor of incision, and then outward through skin flap and back under freed nail margin

Fig 187 —Tying ends of suture automatically invaginates skin flap under nail margin

Fig 188 —Additional sutures permissible in eponychium and at distal end of incision

phalanx, to deformity of the entire nail plate, which on cross section appears horseshoe shaped (Fig 191). Generally, the nail margins invert toward the sides of the distal phalanx Because the contour of the nail largely depends on the contour of the dorsal surface of the distal phalanx, deformity of the nail is often secondary to an anomalous dorsal surface of that bone (Fig 110) It may also be due to hypertrophy of the nail bed

No single surgical procedure can be recommended as applicable to all the variations of the deformity, however, the following procedures give relief in the three general types as outlined

1 When only the margin of the nail is deformed, excision of the nail margin and nail groove, including the matrix of the nail, gives relief This can be performed by the procedures described by Winograd (Fig 176) and by Frost or Steinberg (Fig 177). Dyer and Cohen (1955) make use of negative galvanic current to destroy the groove and matrix after removal of the nail margin

Fig 192 —Frost nail elevator

Regardless of the procedure chosen, it is important that the cells of the matrix under the eponychium be completely destroyed, otherwise spicules of nail may arise from that part of the matrix and grow into the scarified nail groove

2 When the entire nail plate is deformed, Zadik (1950) removes the nail and makes a longitudinal incision on each side of the eponychium, thereby forming a flap of the eponychium The matrix and its bed are excised and the remaining nail bed is elevated distally, the eponychium is sutured to the proximal border of the nail bed

3 DuVries' procedure for avulsion of the nail, denuding the nail bed of hypertrophied tissue, and reducing any dorsal deformity of the distal phalanx is described herewith

(a) Underscore the plate with small Sistrunk scissors or a Frost elevator (Fig 192), and remove it in its entirety This leaves a smooth nail bed surface

not disturbed in the technique During the growth of the new nail, the old nail margin may impinge on the lip because of residual edema secondary to the healing process, however, because its margin is not adherent, a cotton pack can readily be cushioned under it The new nail is not known to have impinged on the nail lip or groove at any time

Panhypertrophy of the nail lips may mask the entire nail (Fig 190,*A*) It may be corrected by excision of a horseshoe section of the hypertrophied lips and suturing of the skin margins (Fig 190, *B* and *C*)

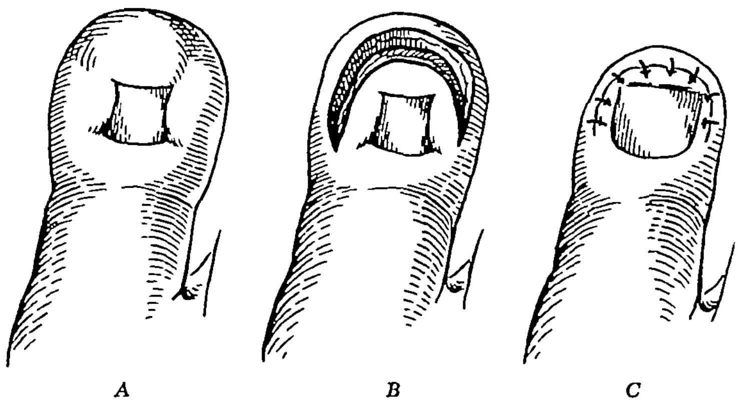


Fig 190 —*A*, Panhypertrophy of lips around nail *B*, Triangular section of skin and subcutaneous fat excised horseshoe-like around toe *C*, Skin margins sutured

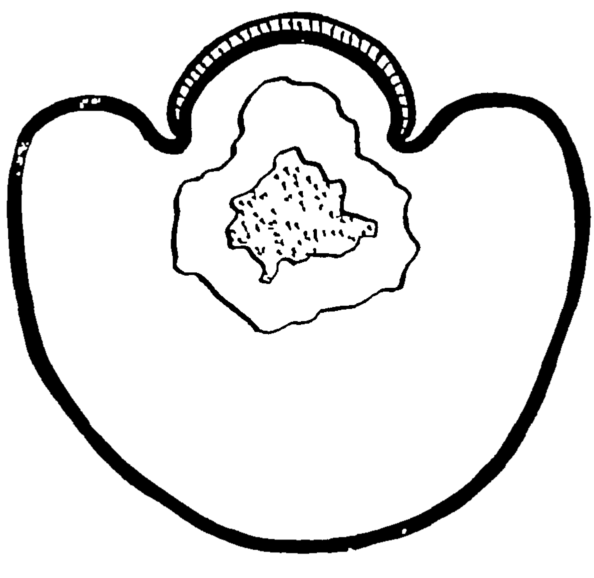


Fig 191 —Incurvation of nail margins

INVERTED NAILS, INCURVATED NAILS; INVOLUTED NAILS

Inverted, incurvated, and involuted nails are ordinarily included in the term *ingrown nail*, comprising about 25 per cent of such cases The disorders are essentially deformities of the nail plate, nail bed, or both The deformities vary widely, from deformity of the nail margins, which curve into the side of the distal

Hyperkeratosis subungualis—Hypertrophy of a nail bed, that is, the tissue beneath the nail plate, associated with onychomycosis, psoriasis, or other dermatoses
Hypertrophied ungualabia—Hypertrophy of the nail lip, associated with so-called ingrown nail
Koilonychia—Concavity in both the longitudinal and transverse axes of the nail plate, caused by nutritional and endocrine diseases
Leukonychia—White spots or striations in the nail resulting from trauma and systemic diseases, such as nutritional and endocrine deficiencies
Onychatrophia—Atrophy or failure of development of nails due to trauma, infection, endocrine dysfunction, or systemic diseases
Onychauxis—Greatly thickened nail plate due to mild persistent trauma and onychomycosis
Onychia—Inflammation of the nail matrix, causing deformity of the nail plate Trauma, infection, and systemic diseases, such as the exanthemas, are causative
Onychitis—Also spelled onyxitis Inflammation of the nail
Onychoclasis—Breaking of the nail
Onychocryptosis—Ingrown toenail
Onychogryposis—Also spelled onychogryphosis. Claw nail or horn nail Extreme hypertrophy of the nail giving the appearance of a claw or horn Condition may be congenital or a symptom of many chronic systemic diseases, such as tinea infections See **onychauxis**, synonym
Onychohelcosis—Ulceration of the nail
Onycholysis—Loosening of the nail plate, beginning in a distal or free edge when trauma, injury by chemical agents, or diseases, such as the acute fevers and syphilis, loosen the nail plate
Onychoma—Tumor of the nail
Onychomadesis—Complete loss of the nail
Onychomalacia—Softening of the nail
Onychomycosis—Fungus infection of the nails, associated with fungus diseases of the feet
Onychonosus—Onychopathy, disease of the nail
Onychophosis—Horny growth beneath toenails
Onychophyma—Thickening or enlargement of the nail
Onychoptosis—Condition in which the nails fall off
Onychorrhexis—Longitudinal ridging and splitting of the nails due to dermatoses, nail infections, systemic diseases, senility, or injury by chemical agents
Onychoschizia—Lamination and scaling away of the nails in thin layers due to dermatoses, syphilis, or injury by chemical agents
Onychosis—Disease or deformity of a nail
Pachyonychia—Extreme thickening of all nails Nails are more solid and more regular than in onychogryposis Usually congenital and associated with hyperkeratosis of palms and soles
Paronychia—Inflammation of tissues about the nail margin Occurs after infection or trauma Infection, either by bacteria or a fungus Injury may be by chemical agents
Polyonychia—The presence of more than one nail on a digit
Pterygium—From the Greek, meaning wing As applied to the nail, term means a thinning of a nail fold and spreading of the cuticle over the nail plate Associated with vasospastic disorders (Word is also referable to conjunctiva of the eye)

REFERENCES

Ashby, B S Primary Carcinoma of the Nail-Bed, Brit J Surg 44 216, Sept, 1956
Bartlett, R W A Conservative Operation for Cure of So-Called Ingrown Toenail, J A M A 108 1257, April 10, 1937
DuVries, H L Hypertrophy of Ungualabia, Chiropody Rec 16 11, Nov, 1933
DuVries, H L Ingrown Nail, Chiropody Rec 27 155-159, Oct, 1944
Dyer, A M, and Cohen, M F A Report on Use of Negative Galvanic Current for Correction of Incurvated Nails, J Nat A Chiropod 45 21, Dec, 1955
Frost, L Root Resection for Incurvated Nail, J Nat A Chiropod 40 19, March, 1950

(b) Make a U-shaped incision with its closed curved end directed distally, following the outline of the nail bed

(c) Denude the dorsum of the distal phalanx of the nail bed, elevating the bed from the bone.

(d) Flatten the dorsal surface of the phalanx with a rasp

(e) Excise all hypertrophied tissue from the undersurface of the nail bed

(f) Replace the nail bed over the phalanx; secure it with a suture through its distal end and the anterior skin lip of the toe

Extensive surgical trauma prolongs healing Drainage may be present for many weeks, requiring frequent dressings Because tinea spores are always present in those regions, fungicides should be applied with each dressing Although a rudimentary or malformed nail usually results, at least the nail is symptom-free

4 A few extremely deformed nails resist all forms of plastic repair, hence, partial amputation of the distal phalanx must be resorted to for removal of the entire nail assembly (For the technical steps, see Chapter 18, Amputations)

SO-CALLED INGROWN NAIL OF LESSER TOES

The lesser toes are subject to diseases and deformities similar to those of the great toe, but they are not so severe and frequent When the primary problem is hypertrophy of the nail lip, the area is smaller and more manageable than in the great toe The distal phalanges of the lesser digits are small bones in comparison with the hallux, they are also comparatively more manageable when the primary problem is one of deformity of the nail The nail lip may be congenitally hypertrophied or become so from restriction of shoes The condition may be remedied by excision of a section of the lip by means of a spindle-shaped incision and suturing of the skin edges

In cases in which the nail bed and plate are deformed, the following procedure is advised

1 Denude the nail from its bed all the way back to the matrix and elevate it

2 Excise all the subungual hypertrophy

3 Underscore the skin over the distal end of the distal phalanx

4 Suture first through the nail at the matrix and out underneath the nail, then pass the needle through the anterior lip, which has been previously freed from the distal phalanx, and then back, pass the needle through the nail again, from its undersurface to its dorsal surface, where the suture is tied to fold the anterior skin lip under the nail, in which position healing takes place

GLOSSARY OF TERMS FOR PATHOLOGIC CONDITIONS OF THE NAIL

Anonychia—Absence of nails, when absence is congenital, usually absence of all nails and condition is permanent Also temporary or permanent loss of one or more nails as a result of general or local diseases

Beau's lines—Transverse lines or ridges marking repeated disturbances of nail growth, caused by wasting diseases or trauma

Hapalonychia—Extremely soft nails that split easily Caused by contact with strong alkalies, endocrine disturbance, or malnutrition

CHAPTER TEN

Disorders of Synovia and Fascia

BURSITIS

THE TERM BURSITIS AS APPLIED TO CONDITIONS IN THE FOOT IS OFTEN MISLEADING, because it implies an inflammation of an anatomic bursa, however, the retrocalcaneal bursa is the only constant anatomic bursal sac in the foot In most people, a rudimentary bursa remains on the tibial side of the great toe joint It contains little or no synovial bursal fluid except when diseased, at which time it forms a ganglion—a pathologic, not an anatomic, entity.

Synovial structures are characteristically lined with endothelial cells and include such tissues as tendon sheaths, joint capsules, pleura, and the peritoneum, as well as anatomic bursae To describe painful areas of the foot as *bursitis* when those areas do not have anatomic bursae is erroneous The tissues referred to in this way are usually joint capsules or formations of adventitious bursae or ganglia. By way of analogy, *myositis* is always understood to mean an inflammation of a muscle, a muscle present in everyone, and no one would apply the term to tissues in which there are no muscles *Bursitis*, on the other hand, has been carelessly applied to areas without anatomic bursae, although synovial membranes are present

Lieberman (1936), for example, described enlarged intermetatarsophalangeal bursae, but apparently he meant *ganglia* The term *bursitis* is often applied to inflammation in various parts of the foot, especially to joints and tendons, where indeed there are synovia but not always normally anatomic bursae, for pathologic or adventitious bursae likewise form in the foot, mostly under the great toe joint, over the fifth metatarsophalangeal joint, over tendon sheaths, and under hard corns Whereas normal bursae are monolocular, pathologic or adventitious bursae are usually multilocular As applied to the foot, therefore, one must be certain regarding the term *bursitis*, which may be an inflammation of

Frost, L Atraumatic Nail Avulsion With a Novel Ungual Elevator, J Am Podiatry A 48.51, Feb , 1958
Gibson, S H , Montgomery, Hamilton, Woolner, L B , and Brunsting, L A Melanotic Whitlow (Subungual Melanoma), J Invest Dermat 29 119-129, 1957
Graham, H F Ingrown Toe Nail, Am J. Surg 6 411, April, 1929
Jansey, F Etiologic Therapy of Ingrowing Toe Nail, Quart Bull Northwestern Univ M School 29 358, 1955
Keyes, E L · Surgical Treatment of Ingrown Toenails, J A M A 102 1458, May 5, 1934
Krausz, C E A Nail Survey of 4600 Patients, J Nat A Chiropod 40 11, May, 1950
Lirch, A O Treatment of Ingrowing Toenail, South Surg 8 173, April, 1939
Maximow, A A , and Bloom, W Textbook of Histology, Philadelphia, 1947, W B Saunders Co , pp 317-349
Newman, R W Simple Treatment of Ingrown Toenail, Surg Gynec & Obst 89 638, 1949
Ney, G C An Operation for Ingrowing Toenails, J A M A 80 374, Feb 10, 1923
Pardo-Castello, V Diseases of the Nails, ed 2, Springfield, Ill , 1947, Charles C Thomas, Publisher, p 3
Steinberg, M D A Simplified Technique for Surgery of Ingrowing Nails, Surgery 36 1132, Dec , 1954
Tauber, E B , Goldman, L , and Claassen, H Pachyonychia Congenita, J A M A 107 29, July 4, 1936
White, C Diseases of Nails, Mississippi Valley M J 61 123-129 July, 1939
Winograd, A M Modification in Technic of Operation for Ingrown Toenail, J A M A 92 229, Jan 19, 1929
Zadik, F R Obliteration of Nail Bed of Great Toe Without Shortening Terminal Phalanx, J Bone & Joint Surg 32B 66, Feb , 1950

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(1) an anatomic bursa, (2) a joint capsule or tendon sheath, (3) an adventitious bursa, or (4) a ganglion

Acute Simple Bursitis

An inflammation of synovia without suppuration, caused by an irritation or injury to a bursa, joint capsule, or tendon sheath, happens mostly over the medial side of the great toe joint, fifth metatarsophalangeal joint, tendo achillis insertion, or the dorsum of the first metatarsal cuneiform. It is induced by ill-fitting shoes

Removal of all irritations and protection of the area by padding gives relief in mild cases. In severe cases, partial or complete immobilization may be necessary

Acute Septic Bursitis

An inflammation of synovia may be caused by an invasion of pyogenic organisms It is often accompanied by pus formation and is usually the result of a wound, although it may be consequent to simple bursitis

Treatment consists of complete rest, hot fomentations, drainage instituted when infection has localized, and prescribed antibiotics When the acute stage has subsided, the patient may be ambulatory, provided pressure has been eliminated from the affected area.

Bursitis From Gout

An acute attack of bursitis is frequently characterized by an acute periarthritis of the great toe joint, often referred to as *gouty bursitis* It has a sudden onset, the great toe joint is extremely swollen and excruciatingly painful The attack is self-limiting but recurs at intervals

Bed rest for three to seven days, hot fomentation over the involved joint, colchicine, and aspirin or cortisone, or both, taken internally, comprise the treatment This therapy shortens the course of the acute stage To prevent acute episodes, the patient should be under the regular supervision of an internist

Chronic Serous Bursitis (Hygroma)

The onset of chronic serous bursitis, sometimes referred to as *hygroma,* is an attack of acute bursitis Continuation of the causative factors makes the condition chronic The synovia becomes thickened and sometimes distended, forming adventitious bursae Occasionally a sinus forms, leading into a joint or into a cavity made by the breakdown of subdermal tissues Chronic serous bursitis affects mostly the tibial side of the great toe joint, a sinus develops which may penetrate into the joint or along the shaft of the metatarsal (Fig 144) The condition also occurs over the phalangeal joints of the lesser toes, where the hygroma may or may not be under a hard corn When the hygroma forms over the tendo achillis, just above the insertion, a sinus may drain into a sac, or the hygroma may be a fibrotic mass

Hygromas should be removed surgically by excising the hypertrophied and distended synovia, including all sinus tracks leading from the bursae When-

ever possible, the entire mass including skin and all surrounding tissue, should be excised, then the skin margins over the area should be sutured All necrotic bone should be curetted out from under the bursa Contraindications for removal, because of the possibility of not healing, are arteriosclerosis, diabetes, and peripheral nerve disease

Tendo Achillis Bursitis

A subdermal enlargement immediately above the tendo achillis insertion is called *tendo achillis bursitis*, although this is not the site of normal anatomic bursae. The mass is likely to be fibrosed and may have a sinus in the center, from which a seropurulent material drains The condition is due to prolonged

Fig 193 —Prominent posterosuperior tuberosity of calcaneus, resulting in massive fibrosis of insertion of tendo achillis

pressure from the upper margin of the shoe counter Any extraordinary shape or a prominent posterosuperior border of the calcaneus (Fig 193) may be the predisposing cause Hohmann (1948) refers to such a prominence as *Haglund's disease* Fowler and Philip (1945) have also called attention to the association of such a prominence with bursitis about the insertion of the tendo achillis The vertical contour of the shoe counter is almost always semielliptical, therefore, when the posterosuperior margin of the heel bone is prominent, the upper margin of the counter constantly irritates the soft tissues over the prominence, producing hypertrophic changes

Excise the fibrotic mass through a longitudinal incision on either side of the tendo achillis The growth is often difficult to excise, because it is contiguous with the skin The skin over the area is devitalized from prolonged trauma which causes the condition in the first place As a result, healing may

be delayed, or there may even be postoperative sloughing It is well to keep the area immobilized for a long period after excision, so that the movement of the ankle will not further traumatize this area If the posterosuperior border of the calcaneus protrudes excessively, which may invite a recurrence of the condition, the protruding part may be amputated and the surface rounded with a rasp

After treatment, complete immobilization with a walking cast should be continued for about three weeks or until healing is well advanced, followed for another month by the wearing of a shoe with the posterior part of its counter removed.

Retrocalcaneal Bursitis

Retrocalcaneal bursitis is an inflammation of the only consistent anatomic bursa of the foot, which is situated between the posterosuperior surface of the calcaneus and the tendo achillis (Fig 35) The condition is usually acute but may be chronic and may or may not suppurate

Etiology.—Friction and pressure from the shoe counter followed by secondary infection are ordinarily causative. Infection may be metastatic Because the bursa is enclosed in a limited area, the infected area is under pressure The symptoms of swelling and inflammation above the posterosuperior portion of the calcaneus, painfulness, and tenderness to touch may be acute, to some degree, acuteness depends on the invading organism. Pain is increased on dorsiflexion of the foot

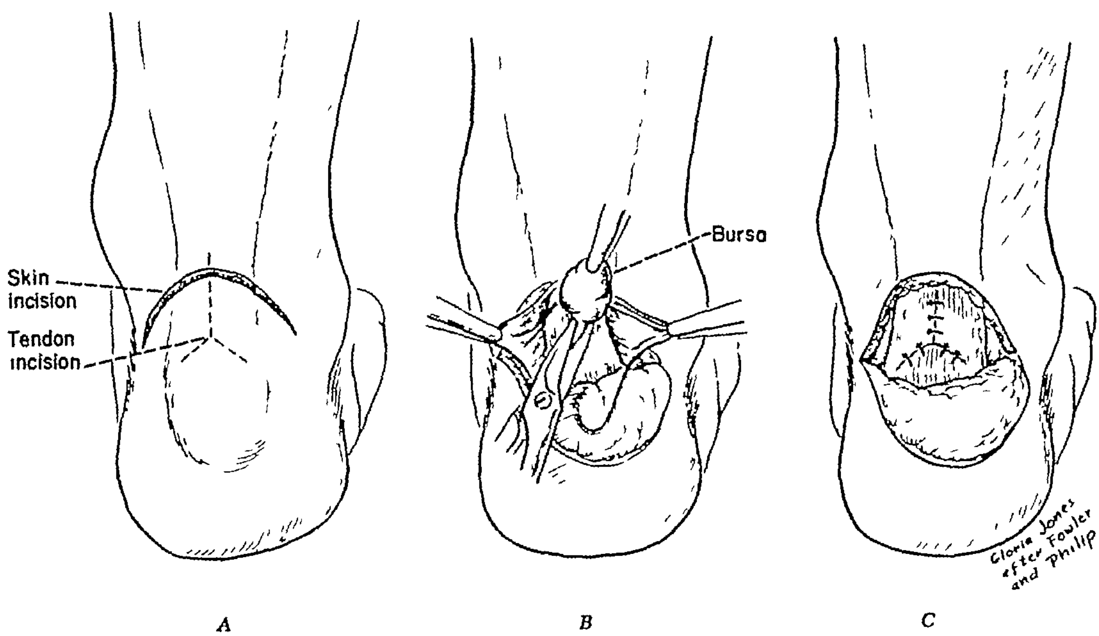


Fig 194.—Fowler and Philip excision of retrocalcaneal bursa A, Semielliptical incision in skin and an inverted Y incision in tendo achillis B, Retrocalcaneal bursa dissected and excised C, Tendo achillis resutured

Treatment.—In the acute stage, treatment is the same as that for acute cellulitis complete rest, hot packs, and antibiotics If the infection becomes organized, drainage should be instituted In chronic or recurrent cases, the bursa needs to be excised, however, such excision is at times followed by lengthy and painful convalescence, because of the difficulty of occluding the dead space left by removal of the bursa A hematoma may form in that space

Technique by Fowler and Philip—A mild convex incision with its vertex upward is made over the tendo achillis about 1 cm above the posterosuperior lip of the calcaneus The skin margins are freed and retracted A vertical incision about 6 cm in length is made midline in the tendo achillis At the base of the incision, the tendon is incised for about 1 cm on each side of the midline in an oblique downward and outward direction to form an inverted Y (Fig 194) The tendon flaps are retracted, and through the aperture the bursa is excised and the bony prominence of the calcaneus is removed with an osteotome and rounded with a rasp The tendon is repaired with interrupted sutures, and the skin is closed A plaster leg cast with a walking caliper is applied The foot is held in mild equinus position This technique may leave a scar, which the shoe irritates The recommended technique does not leave a scar

Recommended Technique in Treatment of Retrocalcaneal Bursitis—The steps are as follows

1 Make a longitudinal incision about 4 cm long over the tibial side of the retrocalcaneal space (Fig 195)

2 Retract the skin margins to expose the retrocalcaneal bursa

3 By holding the foot plantar flexed, the tendo achillis is readily deflected to facilitate the approach to the bursa

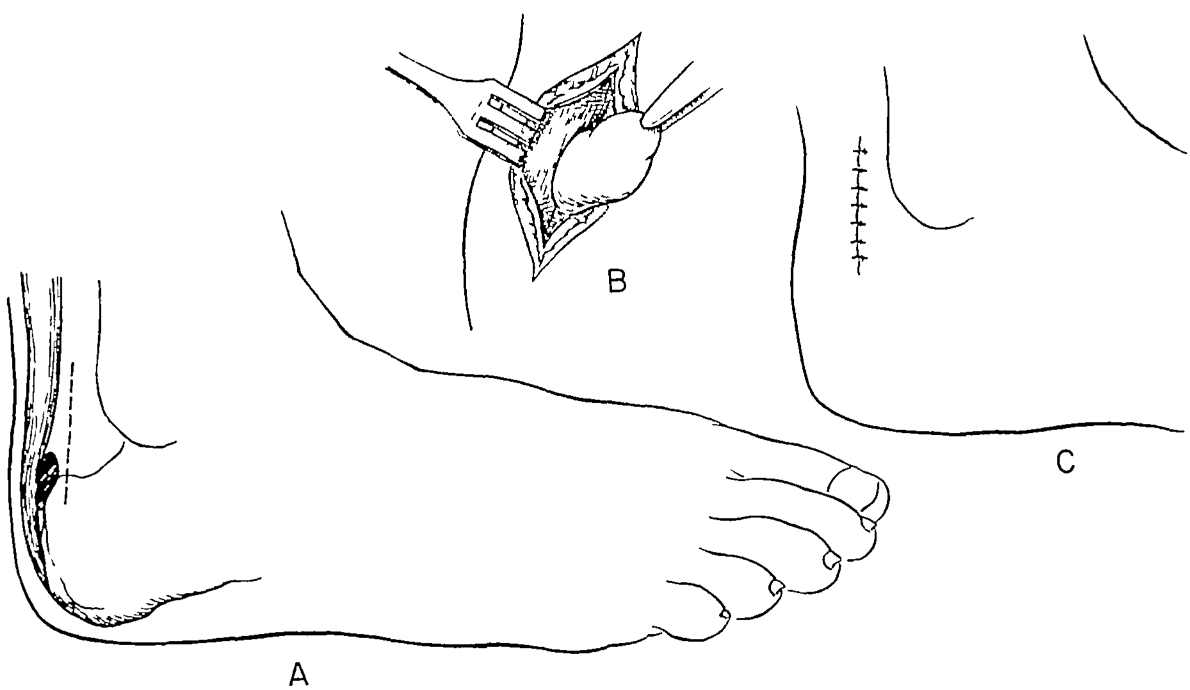


Fig 195—*A*, Incision along lateral side of terminal end of tendo achillis *B*, Retrocalcaneal bursa dissected and excised while foot is held in plantar flexion *C*, Skin and fascia sutured

4 Dissect and excise the bursa, which is usually fibrosed

5 Suture the skin with fine catgut subdermally to the opposite side so as to obliterate the dead space, then suture the skin incision

6 If the subdermal sutures do not obliterate the dead space, place a button on each side of the retrocalcaneal space and carry a wire suture with a Keith needle through the buttonholes and the space

7 Apply a leg cast with walking heel, with the foot held in mild equinus

Postoperative Treatment—Twenty-four hours after the operation, make a window in the cast over the retrocalcaneal space to allow for swelling If a button suture has been used, it may be removed on the second postoperative day, because by then any danger of formation of hematoma will have passed

Adventitious Bursae Under First Metatarsal Head

An adventitious bursa under the first metatarsal head is an accumulation of lobular bursal tissue under the great toe joint The accumulation often reaches massive size (Figs 196 and 197). It is observed most in girls during adolescence or in early adult life It is due to a chain of conditions, such as hypermobile sesamoids, sesamoids that are articulate immediately under the great toe joint space, and bifurcated tibial sesamoid, especially if that bone is distorted or abnormal in size These factors, in combination with the wearing of high-heeled shoes before the sesamoids have completely ossified, and excessive dancing and pivoting on the first metatarsophalangeal joint cause the defensive formation of a bursa On palpation, the mass feels like a large bulbous structure under the skin, which is freely movable and compressible

Treatment by Recommended Technique for Removal of Adventitious Bursa Under First Metatarsal Head.—Excision of the mass is indicated If the tibial sesamoid is dislocated, hypertrophied, or anomalously shaped, it should likewise be excised

1 Make an incision following the curve of the medioplantar border of the great toe joint

2 Retract the skin margins and free the plantar flap from the entire plantar surface of the great toe joint Retract as far as possible

3 Expose the bulbous mass, dissect, and excise

4 If, on palpation of the tibial sesamoid, sharp points are felt or the shape on the plantar surface is anomalous, the tibial sesamoid should be excised Make a horizontal incision in the tibial side of the metatarsophalangeal capsule at the juncture of the superior surface of the tibial sesamoid with the inferior surface of the head of the first metatarsal (See Chapter 12, page 257)

5 Grasp the medial margin of the sesamoid, free its entire attachment with a scalpel and enucleate the bone completely

6 Close the capsule with interrupted catgut sutures, then close the skin

7 Apply a compression bandage Ambulation may begin in forty-eight hours

Postoperative Course—Postoperative pain is mild Sutures should be removed in from nine to eleven days A dressing which will partly immobilize the great toe joint should be maintained for four weeks, at which time full weight bearing may be resumed

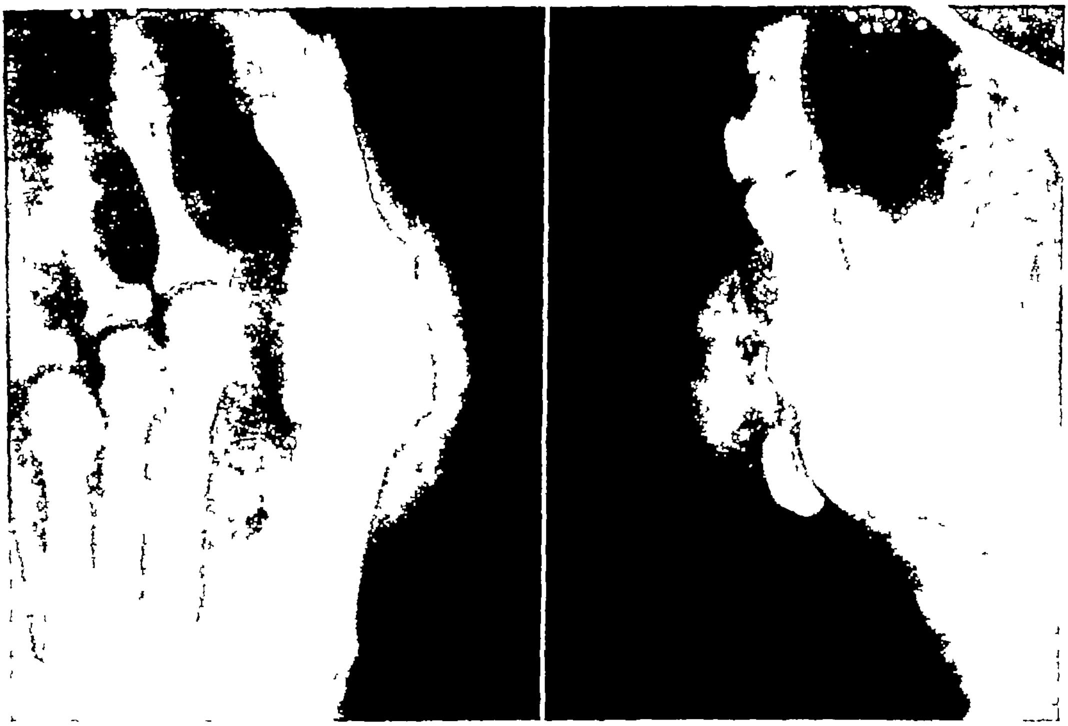


Fig 196—Dorsiplantar and medial view of large adventitious bursa under first metatarsophalangeal joint

Fig 197—Contrast dye injected in adventitious bursa under first metatarsophalangeal joint

Management of Adventitious Bursae Under Other Weight-Bearing Surfaces.—A comparable adventitious bursa sometimes forms as a protection against excessive pressure under any other weight-bearing surface, such as the plantar surface of the lesser metatarsal heads or the tuberosity of the calcaneus Malformation, deformities, or anomalies of those bones induce formation of adventitious bursae Such bursae vary from small to large proportions, depending on the cause The plantar surface of the fifth metatarsal is the next likely area where they may form (Fig 198). Excision of the bursa, correction of the causative deformity, and prevention of excessive pressure under the area through proper weight distribution resolve the problem

Fig 198 —Large adventitious bursa under fifth metatarsophalangeal joint

Adventitious bursae under a corn are multilobular masses filled with a synovial-like fluid, frequently encountered under helomas They are formed as a protection against pressure on a bony prominence, however, they increase that irritation inasmuch as the original cause of the pressure persists Usual sites are on the fifth toe or over a hammered toe of any of the three middle toes On the fifth toe, they may be excised through a dorsal longitudinal incision at the same time that the offending condyle is excised (See Heloma on Fifth Toe Operation, page 172) Over a hammered toe, the skin and the bursa may be excised through a transverse elliptical incision (See Fifth Toe Hammertoe Operation, pages 354 to 356)

GANGLION

The word *ganglion* is a term describing a certain growth which may occur about the joints or tendons of the extremities

Etiology.—The growth, which never becomes malignant, apparently arises from degeneration in the connective tissue outside of the joints (King, 1932) Ganglia were formerly thought to be cysts that arise from aberrant islands of synovial tissue or from herniations through the linings of joints or tendon sheaths,

Fig 199

Fig 200

Figs 199 and 200 —Ganglion on medial side of first metatarsophalangeal joint

but inasmuch as they contain neither synovial endothelium nor synovial fluid, that concept has been invalidated. It is likely that they are caused by mucoid degeneration of connective tissue collagen, probably consequent to trauma (Fisk, 1949). A ganglion is a lobular mass over a joint capsule or tendon sheath, often over a bony prominence, and contains gelatinous material which is usually colorless but may be straw colored. It rarely penetrates the deeper structures, Fisk, however, did report such a case. Ganglia vary in size from 0.5 cm. or smaller to 8.0 cm. Ganglia are freely movable and cause no pain except when pressure is exerted on them.

Fig. 201.—Ganglion over dorsum of first cuneiform.

Ganglia occur on the foot in the following order of frequency: medial side of the great toe joint (Figs. 199 and 200), dorsum of the first metatarsocuneiform joint (Fig. 201), over any of the extensor tendons, lateral side of the fifth metatarsophalangeal joint, or in a metatarsal interspace. In a metatarsal interspace, ganglia are often multilocular and have ducts which lead to other sacs in the plantar fascial planes. The largest part of the body of a ganglion may be present in the web between the toes, where it begins and continues proximally between the metatarsal heads and then escapes into the plantar fascial spaces. In Fisk's case, the ganglion caused a massive bone concavity of the medial malleolus and lower end of the tibia.

Conservative Treatment.—Palliative relief may be given by aspirating the cavity, however, the cavity usually refills in a few days. Attempts to eradicate the ganglion by aspirating the contents and injecting a sclerosing material have not been encouraging. Because there is no method of controlling the drug once

it has been instilled into the growth, it may not be capable of destroying the lining of the ganglion sufficiently, or the result may be sloughing of the entire area

Surgical Treatment. Recommended Technique for Removal of Ganglion.—Surgical excision of the ganglion is the most successful treatment An incision is made immediately over the mass If the ganglion is under a weight-bearing surface, the incision is made on the side of the foot closest to the growth, and the ganglion is gently freed from the surrounding tissue During dissection, the sac may rupture, expelling its contents into the wound In such cases, the sac should be grasped, its course followed to its origin, and excised It may be difficult to follow the course of a ganglion that occurs in a metatarsal interspace This may necessitate an extended incision, or a second incision, in order to extirpate the entire ganglion If a small lobe or its source is allowed to remain, the condition will recur All dead spaces left by removal of the ganglion must be obliterated by subdermal suturing A compression bandage is then applied

FASCIAL HERNIA

An area of the fascia of the foot may rupture so as to permit a protrusion of the soft subfascial tissues This may happen on the dorsolateral aspect of the foot, especially over the tarsal bone, where the extensor brevis digitorum muscle may herniate The rupture is likely to be longitudinal and vary in length from 0 5 to 4 cm It may simulate a ganglion or lipoma Fascial hernia may or may not produce symptoms

The rupture can be repaired by coaptating the margins of the ruptured fascia and suturing them In rare instances, the margins cannot be approximated In such cases, a piece of fascia lata may be sutured over the hernia

DUPUYTREN'S CONTRACTURE (FIBROMATOSIS OF THE PLANTAR FASCIA)

In 1832 Dupuytren first described the clinical entity that bears his name as a fibrosis of the palmar fascia The lesion is characterized by the invasion of fibromatosis in the *palmar* fascia (Pedersen and Day, 1954) It has received more attention (Meyerding and Shellito, 1948) than the comparable *plantar* fascia involvement (Fig 202) As Pickren and his associates (1951) have pointed out, involvement of the plantar fascia may not be recognized, because contraction of the toes does not take place, and therefore the involvement is often erroneously diagnosed as fibrosarcoma It is apparent that such a fibromatosis of the plantar fascia is more prevalent than is generally realized

Allen and his associates (1955) reviewed the published reports and presented the results of their study of sixty-nine additional cases of plantar fibromatosis and nine cases of malignant neoplasms of the soft tissues of the sole Goetzee and Williams in the same year reported a case of a child who had such contractures on both hands and feet In one of my cases, the growth occurred on both feet of a man, aged 34 years The mass was on the medial border of

the plantar fascia under the base of the first and second metatarsals On the right foot the mass measured 6 by 4 by 3 cm (Fig. 203). The growth on his left foot was somewhat smaller The masses were the highest area of the plantar surface, which forced the patient to walk on the outer borders of his feet in extreme inversion.

Etiology.—The cause in most cases is obscure. Skoog (1948), in his review of published reports, listed the following general conditions as probable causes injury, neuropathy, gout, rheumatism, endocrinopathy, local infection, and tuberculosis

Fig 202 —Fibromatosis of plantar fascia in an adolescent

Symptoms.—Pain is felt on weight bearing The patient is aware of the presence of a new growth, which may be the highest area of the weight-bearing surface of the foot The longitudinal arch loses elasticity The accumulation of the fibrotic mass may be massive

Pathology.—The gross change is essentially a nodular thickening of the plantar fascia which varies in size and shape and usually adheres to the overlying skin Microscopically, the changes consist of islands of proliferating fibroblasts, which may be mistaken for a malignant neoplasm because of their invasive properties (Allen and co-workers, 1955) Invasiveness takes place especially after previous surgical excision

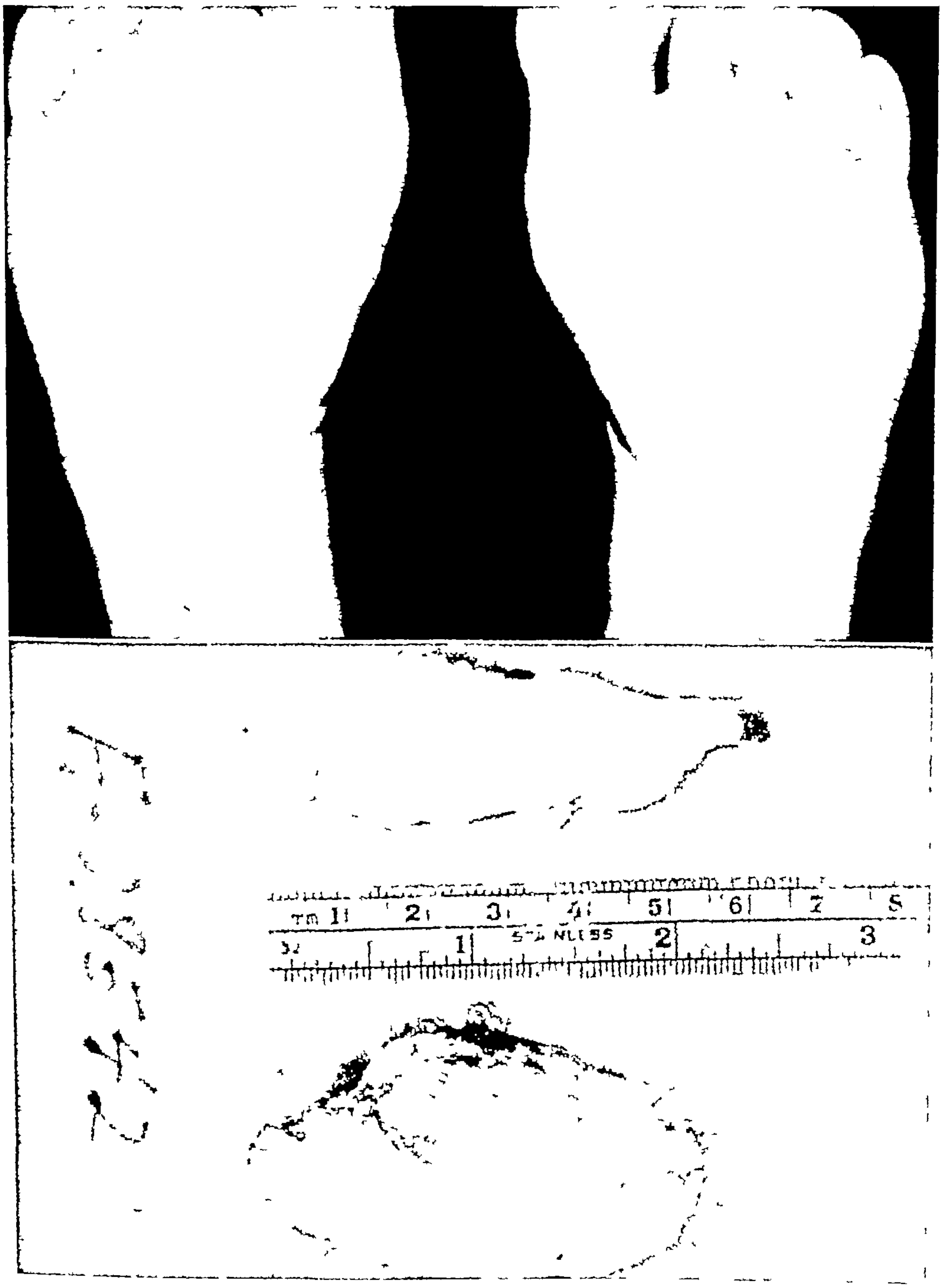
Technique for Excision of Dupuytren's Lesion of the Plantar Fascia—Pedersen and Day (1954) admonished that unless the entire mass is completely excised, the disease tends to recur

1 Make an incision along the medioplantar border of the first metatarsal shaft extending to the navicular

2 Retract the skin, exposing the plantar fascia.

3 Completely excise the mass, which incorporates the fascia in most instances.

A



B

Fig 203—*A*, Recurrent massive fibromatosis (Dupuytren's contracture) on both feet *B*, Gross specimen excised *C*, Postoperative appearance

4. If a large area of the plantar skin has to be freed, it should be sutured with subdermal catgut to the remaining tissue so as to avoid formation of a hematoma.

5 A compression bandage restricts subdermal bleeding Release the compression in twenty-four hours, and permit the patient to begin mild weight bearing on the fifth or sixth postoperative day

C

Fig 203 (cont'd) —For legend see page 235

VOLKMANN'S ISCHEMIC CONTRACTURES

Contraction of one muscle or a group of muscles due to the displacement of the muscle cells by connective tissue is caused by an interference with the blood supply to the muscle involved The condition is known as *Volkmann's ischemic contractures* A tourniquet or cast, especially over the arm, may cause the contraction by injury to a major artery Trauma may be directly to the artery supplying the muscle or to a distal portion of the vessel, resulting in reflex vasospasm along its course through the leg The foot is affected only indirectly

In the early stage the loss of blood supply produces necrosis (muscle sequestrum) in the body of the muscle In time, fibrous connective tissue replaces dead muscle fibers The fibrous connective tissue contracts and shortens, thereby producing a deformity of the limb

Treatment is primarily directed toward overcoming the deformity, for reviving the muscle fibers is unlikely Treatment consists of prolonged stretching, tendon lengthening, or sectioning of the contracted muscle,

REFERENCES

Allen, R A, Woolner, L B, and Ghormley, R K Soft-Tissue Tumors of the Sole, With Special Reference to Plantar Fibromatosis, J Bone & Joint Surg 37A 14-26, Jan, 1955

Fisk, G R Bone Concavity Caused by a Ganglion, J Bone & Joint Surg 31B.220-221, May, 1949

Fowler, A, and Philip, J F Abnormality of Calcaneus as Cause of Painful Heel, Brit J Surg 32 494-498, April, 1945

Goetzee, A E, and Williams, H O A Case of Dupuytren's Contracture of the Hands and Feet in a Child, Brit J Surg 42 417-420, Jan, 1955

Hohmann, G Fuss und Bein, Munchen, 1948, Verlag von J F Bergmann, p 299

King, E S J Pathology of Ganglion, Australian & New Zealand J Surg 1 367-381, March, 1932

Lieberman, H S Enlarged Intermetatarsophalangeal Bursae of Both Feet, J Bone & Joint Surg 18 1062, Oct, 1936

Meyerding, H W, and Shellito, J G Dupuytren's Contracture of the Foot, J Internat Coll Surg 11 595-603, 1948

Pedersen, H F, and Day, A J Dupuytren's Disease of the Foot, J A M A 154 33, Jan 2, 1954

Pickren, J W, Smith, A G, Stevenson, T W, Jr, and Stout, A. P Fibromatosis of the Plantar Fascia, Cancer 4 846-856, July, 1951

Skoog, Tord Dupuytren's Contraction, With Special Reference to Aetiology and Improved Surgical Treatment, Its Occurrence in Epileptics, Note on Knuckle-Pads, Acta chir scandinav (supp 139) 96 1-190, 1948

CHAPTER ELEVEN

Disorders of Tendons

THE OBJECTIVES OF SURGICAL PROCEDURES APPLICABLE TO THE TENDONS ARE TO reduce deformity, repair injury to a tendon, and improve function by repairing, replacing, or transferring tendons The changes in tendons produced by disease or deformity differ from those in less specialized tissue The gliding mechanism of a tendon differs according to whether it pulls around an angle or in a straight line (Figs 204 and 205) The first type is covered with tendon sheath and fibrous bands which are adherent to the periosteum of the bone The second type is covered with paratenon, a highly specialized elastic fat, the tendon is adherent to the paratenon which moves with it during motion in the manner of an accordion The paratenon is instrumental in healing of the tendon and aids in preventing scar formation

The tendon sheath is made up of two layers of synovial membrane, the inner layer covers the tendon and the outer layer forms a lining for the canal in which the tendon glides When the paratenon is destroyed and the tendon sheath is greatly injured the sheath becomes adherent to the tendon and to adjacent skin and bone, so that if the tendon pulls around an angle, deformity results If the circular fibers are also destroyed, the tendon may pull out and act as a bowstring

HEALING OF TENDONS

When a tendon is sectioned and the cut ends of a tendon are held in proximity to each other, not permitting the proximal end to be pulled away by muscle spasm, the ends undergo healing for four weeks, similar to wound healing of other soft tissues which takes about four weeks to become firm enough to withstand tension or traction During the first week a gelatinous substance forms around the severed ends, comparable to the stage of thrombosis in the healing of soft tissue During the next ten days an influx of fibroblasts and collagen fibers

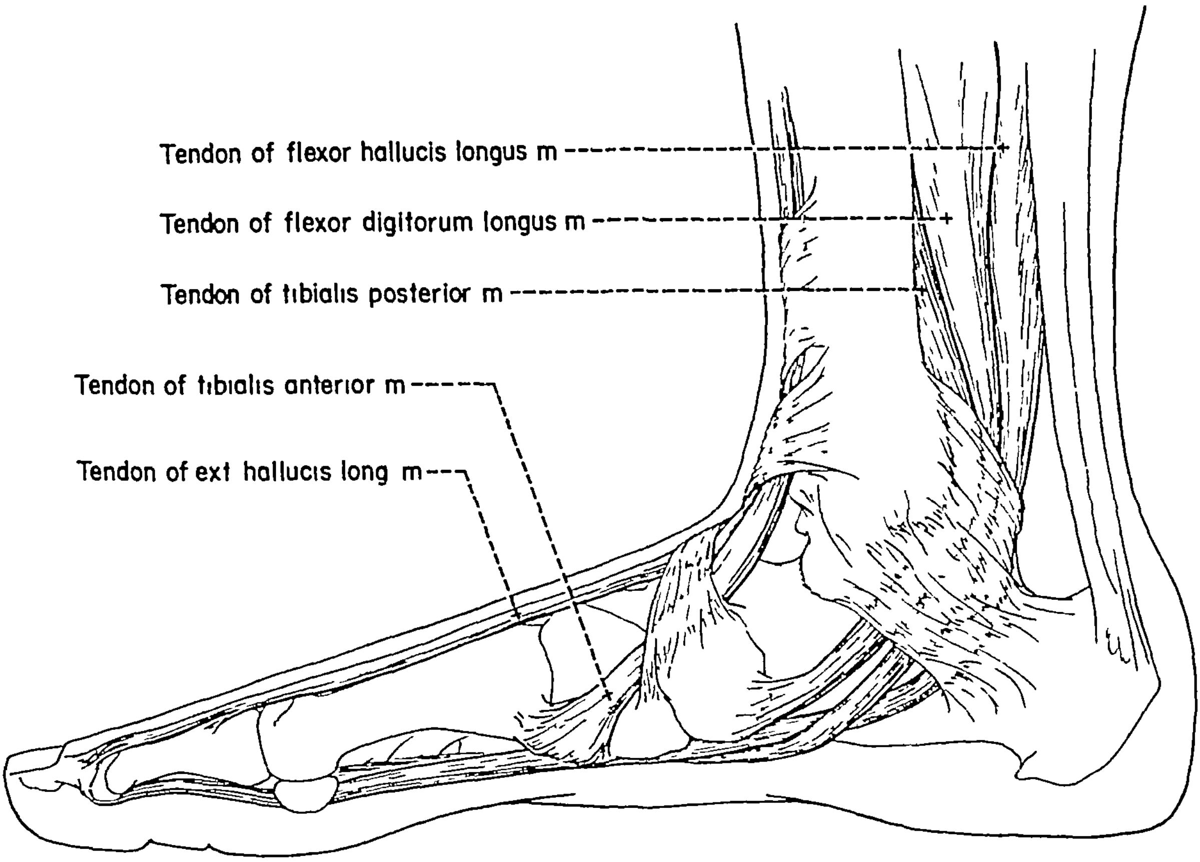


Fig 204 –Medial view of course of tendons of foot and ankle

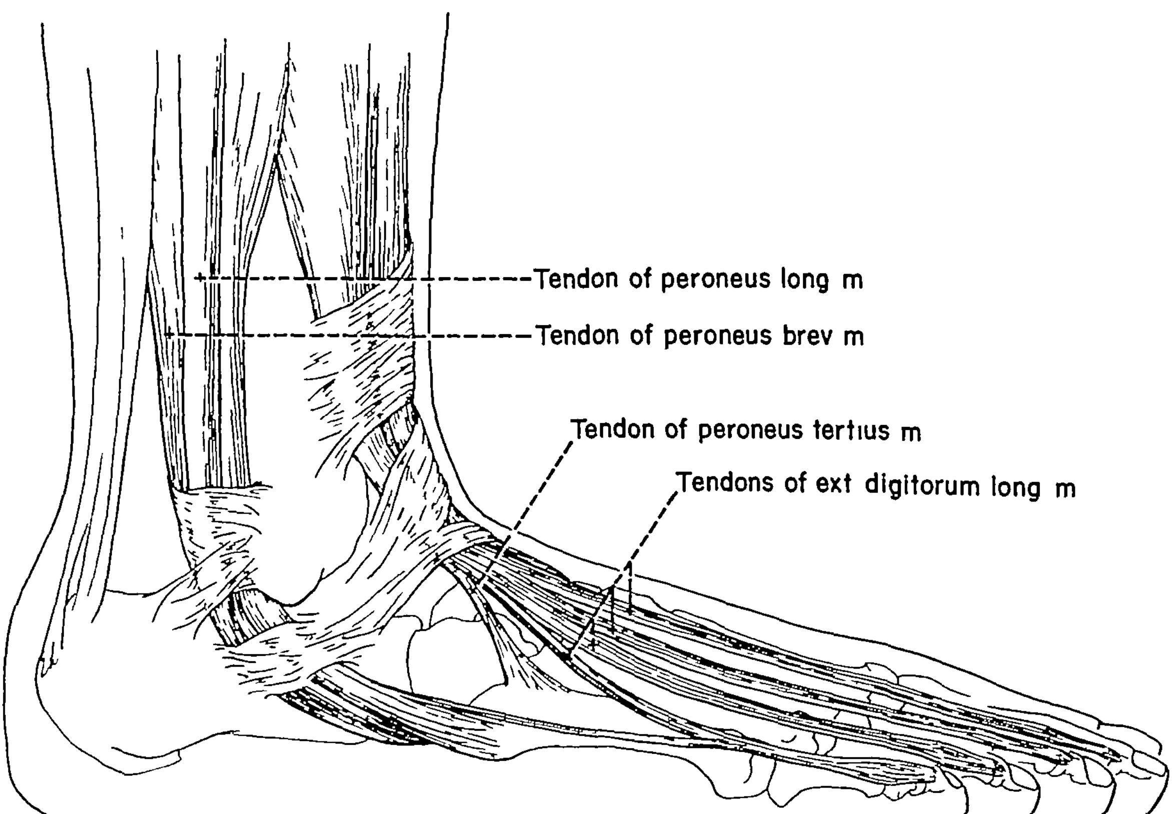


Fig 205 –Lateral view of course of tendons of foot and ankle

strengthen the area, comparable to the stage of fibroplasia in other soft tissues During the next two weeks healing progresses, small arterioles recanalize, and union becomes firm enough to withstand movement Absorption of the healing elements takes place This permits the tendon to loosen from the surrounding tissue, which restores free movement of the tendon

Healing elements refer to dead tissue which is always present after tissue has been injured surgically or accidentally The reference is also to the altered tissue from thrombotic and inflammatory changes, all of which combine to alter the area into a solid mass Those elements must be absorbed before any of the tissues in the area can assume their normal excursions This is true of the healing process of most soft tissues

Tendons of the foot do not perform the delicate function performed by those of the hand, therefore, their repair requires somewhat less meticulous surgical procedure However, the mechanics of the two organs and their pathologic conditions are similar Bunnell (1956) has contributed greatly to our understanding of the function and structure of the tendons of the hand That knowledge is largely applicable to the subject of surgery of the tendons of the foot

INFECTION AND INJURY

Bacterial Tenosynovitis

Bacterial tenosynovitis is an inflammation of tendon sheaths, which may be suppurative or nonsuppurative It is always due to bacterial invasion of the involved tendon or tendons The course of the disease depends largely on the invading organism and on how it invades the tendon sheath

Acute Infectious Tenosynovitis

Acute infectious tenosynovitis (Christie, 1955) is due to spread of infection from adjacent tissues, from direct inoculation by accidental laceration, or by contamination from an incision made because of a subcutaneous infection The laceration or puncture may be microscopic The infection may also spread from other parts of the body

The invading organism in most cases is one of the common pyogenes, although infection with other organisms has been reported The symptoms are the classic signs of inflammation along the course of the tendon, producing extreme pain, especially at the insertion of the tendon, even though the active infection may be at a distance This is Nature's attempt to immobilize the infected tendon Any tendon and its sheaths may be involved, but the infection is commonest in the extensor tendons of the foot Sloughing of the tendons is a complication which may lead to extreme contracture

Treatment consists of complete rest of the foot and prompt administration of chemotherapy Massive hot fomentations should be applied continuously night and day Only in rare cases should drainage be instituted by incision and then only when fluctuation gives evidence of organized pus Serious injury to the gliding mechanism may be caused by incising a tendon that does not contain purulent material It is better to err on the conservative side of treatment

Chronic Infectious Tenosynovitis

Chronic infectious tenosynovitis is due to a specific disease, such as syphilis or tuberculosis. It may be the only manifestation of active tuberculosis. Chronic infectious tenosynovitis is rare, but when it occurs, it involves the sheaths of the extensors and peroneal tendons around the ankle joint.

Treatment consists of immobilization of the affected tendons and general treatment of the underlying systemic disease. Surgical repair of the gliding mechanism may be necessary when injury to the tendon and its sheath is severe.

Traumatic Tenosynovitis

Traumatic tenosynovitis may be acute or chronic.

Acute Traumatic Tenosynovitis.—The acute type is due to sudden trauma by extreme stretching of one or more tendons as may happen during strenuous exercise. The tendons involved most are the long extensor of the great toe, the peroneus longus, and the tendo achillis, although any long tendon may be affected. The symptoms are sudden pain and swelling of the injured tendon.

If the patient is seen within twenty-four hours after injury, the limb is put at rest under cold applications. If seen later, heat is applied for forty-eight hours, followed by immobilization for two weeks to a month, depending on the degree of injury. In mild cases adhesive strappings suffice, in severe cases a plaster cast with a walking caliper is required. Severe cases and cases of repeated traumatic tenosynovitis may produce a great deal of scarification and freeze the tendon, requiring extensive repair and the formation of a new gliding mechanism.

Chronic Traumatic Tenosynovitis.—The chronic type may result from repeated trauma to the same tendon and may produce a formidable problem. The tendon may become limited in its course of movement, because of scarring of the tendon sheath. Constant pain and impairment of function of the particular muscle result. The chronic type may also be due to pressure and friction of the shoe over a prominent tendon. In rare instances the condition results from foot imbalance which strains one or more tendons of the leg muscles. These patients show improvement once the imbalance has been corrected.

Those injuries due to improper footgear generally involve the extensor hallucis longus or the tendo achillis. The extensor hallucis longus is most prominent over the dorsum of the forefoot. This is why it is often impinged upon by the margins of the vamps of pumps. Sometimes a protective fibroma of the skin and subcutaneous tissue forms just behind the first metatarsal head, which may cause recurrent infection and formation of a draining sinus in its center. The entire mass of scar must be excised and at times the gliding mechanism must be repaired as described on page 199.

A comparable condition is observed just above the insertion of the tendo achillis which also results from friction and pressure—in this instance of the shoe counter. This is discussed on page 199 and under Fibromatosis of the Tendo Achillis (pages 225 and 226).

Hemorrhagic Tenosynovitis

Hemorrhagic tenosynovitis is an uncommon complication of an injury to a tendon A direct blow to the tendon or an excessive strain of the tendon may break the endothelial lining of the tendon sheath, causing hemorrhage into the sheath, from which it cannot escape and therefore clots The blood clots assume the character of a foreign body, thereby becoming an irritant and often producing muscle spasm This accounts for the continuous pain, even when the limb is at rest The irritation tends to induce an increase in the production of synovial fluid in the sheath, causing fluctuation over the area

Acute pain and swelling over the injured tendon begin from twenty-four to forty-eight hours after trauma Pain grows worse and cannot be relieved, rest or change in position of the limb does not modify the symptoms The area over the tendon is swollen, fluctuant, and tender to touch. The condition is differentiated from a pyogenic tenosynovitis by its rapid onset of symptoms after injury, the absence of signs of infections, and persistent, violent, and unrelenting pain over the injured tendon, which are incompatible with simple injuries to a tendon

Exquisite pain and fluctuation pinpoint the site of the hematoma, which must be excised through an incision in the tendon sheath Only rarely is the clot absorbed without excision.

TRAUMA TO SPECIFIC TENDONS

Lapidus and Seidenstein (1950) classify chronic nonspecific tenosynovitis into (1) peritendinitis crepitans, (2) stenosing tenosynovitis, and (3) chronic tenosynovitis with effusion They reported three cases, two involving the posterior tibial tendon and one, the flexor hallucis longus Pain was intermittent in all cases The condition had remained unrecognized for several months in the case involving the flexor hallucis longus and for one and three years, respectively, in the cases involving the tibialis posticus The tendon sheath was opened to evacuate the excess fluid, and the thickened part of the sheath was excised

Burman (1953) reported sixteen cases of stenosing tendovaginitis (tenosynovitis) of the foot and ankle treated by surgical repair of the gliding mechanism Most cases concerned the extensor tendons, but the causes differed

Ghormley and Spear (1953) reviewed twenty-one cases of tenosynovitis of the tibialis posticus diagnosed and treated at the Mayo Clinic between 1935 and 1951 Ten patients were treated conservatively with roentgen therapy and strapping, and the remaining eleven received surgical treatment Some cases were due to anomalies of the tibialis posticus The variations were essentially accessory posterior tibial tendons which were excised at the time of operation

Cohen and Reid (1935) reported seven cases of tenosynovitis crepitans associated with oxaluria

TRAUMATIC SUBCUTANEOUS RUPTURE OF TENDONS

Traumatic avulsion of tendons of the foot is uncommon Those cases reported are mainly of the tibialis anticus Burman (1956) reported a case of subcutaneous rupture of the peroneus longus tendon

Els (1910) was one of the first to report a rupture of the *anterior tibial tendon*. Burman (1934) was first to report such a case in America. Lapidus (1941) reported two cases Mensor and Ordway (1953) reported two cases and reviewed the published reports, of which only ten had appeared up to that time. Lipscomb and Kelly (1955) reported twelve cases of injuries to the extensor tendons of the foot treated at the Mayo Clinic between 1938 and 1952, in nine of the cases the tibialis anticus was involved.

Trauma causing avulsion may be powerful or mild Usually it is caused by a fall or twisting of the ankle, especially when extreme plantar flexion is

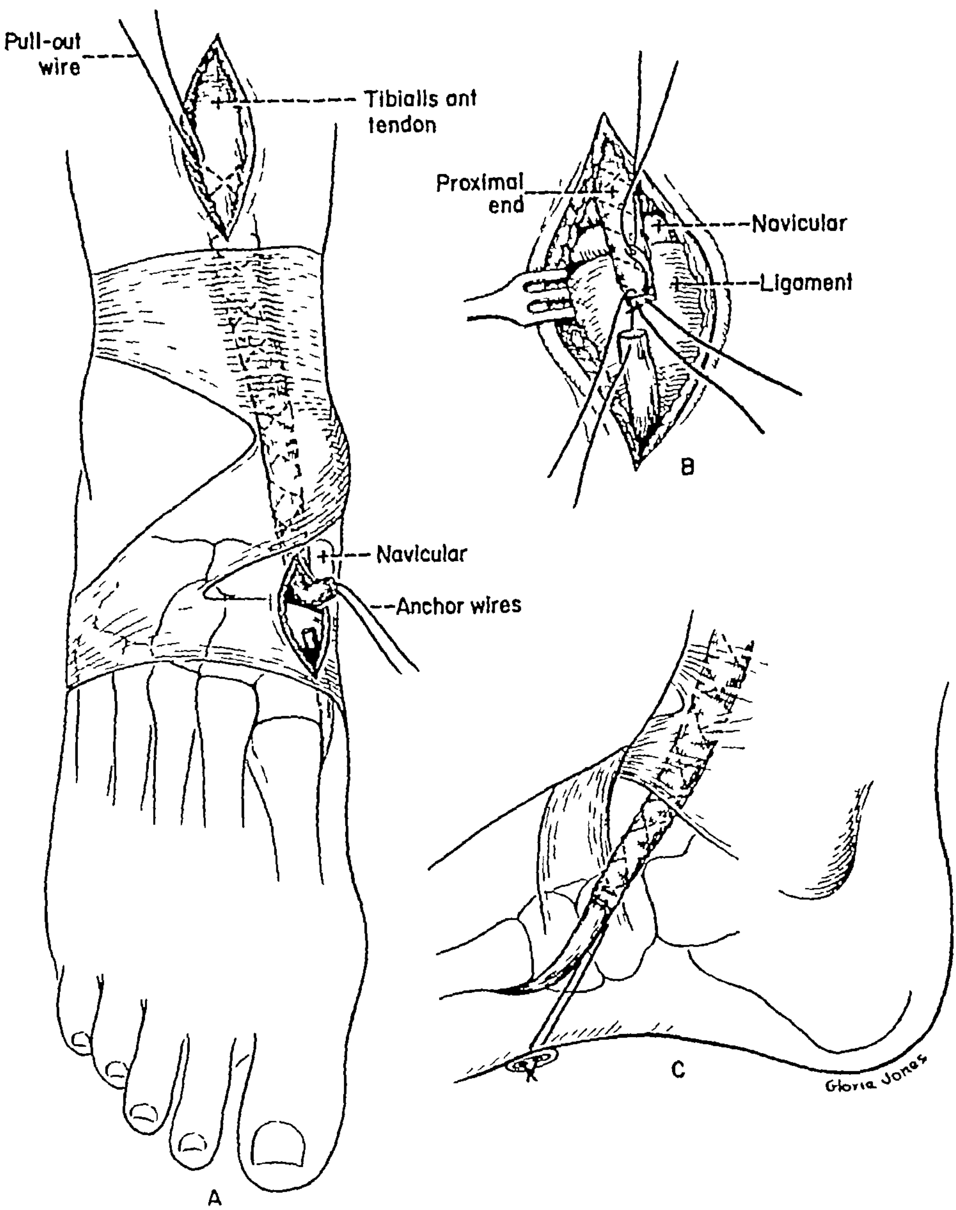


Fig 206—Repair of ruptured tibialis anticus tendon *A*, Incision over tibialis anticus tendon, just above transverse crural ligament Second incision over insertion of tibialis anticus Tibialis anticus tendon pulled out through first incision Stainless steel wire threaded through tendon from above downward At proximal end a pull-out wire is looped under threaded wire and the tendon guided through its original channel *B*, Fragmented ends sutured to each other *C*, Both ends of stainless steel wire passed through plantar surface by means of Keith needle and tied to a button

exerted Sometimes the same area has been previously injured, probably with residual fraying of the terminal end of the tendon, predisposing it to complete rupture

Symptoms.—*Symptoms of ruptured tibialis anticus tendon are sudden sharp* pain and swelling over the first cuneiform, accompanied by an inability to coordinate normal foot motion Dorsiflexion of the foot may be difficult, and there may be a tendency to stub the toes A few days after injury, extensive ecchymosis may form over the insertion of the tendon On palpation, a rupture defect is observed in the tendon channel at the terminal end of the tibialis anticus When edema subsides, the defect may become visible The terminal end of the ruptured tendon feels like a bulbous mass

Recommended Treatment.—To repair a ruptured tibialis anticus, the following steps are performed

1 Make an incision over the tibialis anticus tendon just above the transverse crural ligament

2 Make a second incision over the insertion of the tibialis anticus tendon

3 Pull out the tibialis anticus tendon through the first incision and thread a stainless steel wire through the tendon from above downward At the proximal end of the threaded wire, loop a pull-out wire

4 Replace the wired tendon into its original tunnel

5 Suture the frayed ends of the tendon to each other and to the periosteum and cortex of the first cuneiform

6 The stainless steel wires are passed through the plantar skin surface by means of Keith needles and tied to a button (Fig 206) Bunnell (1956) may be credited with being the first to use the button innovation A boot cast is applied for six weeks After its removal the threaded wire is cut at the button, the pull-out wire permits extraction of the threaded wire

OSSIFICATION OF TENDO ACHILLIS

Ossification of the tendo achillis is a distinct, although rare, clinical entity Horing (1908) and Jacobsthal (1909) were among the early writers to call attention to this disease Ghormley (1938) reviewed the published reports of twenty-one cases, of which sixteen were in men In eighteen instances the ossification developed free in the tendon, and in three, it was an outgrowth of the posterior tuberosity of the calcaneus In many of the patients the tendon had previously been traumatized

The cause of the disease is obscure, many conjectures have been offered, such as causation by (1) myositis ossificans, (2) a neoplasm, (3) an accessory sesamoid None fits into a rational pattern indicating why ossification occurs only in this tendon of the foot, the tendo achillis Extensive ossification (Figs 207 and 208) of the body of the tendon is rare, but ossification at the insertion of the tendon (Figs 209 and 210) is common The only consistent symptom is limitation of motion of the ankle, the degree of limitation depending on the extent of ossification

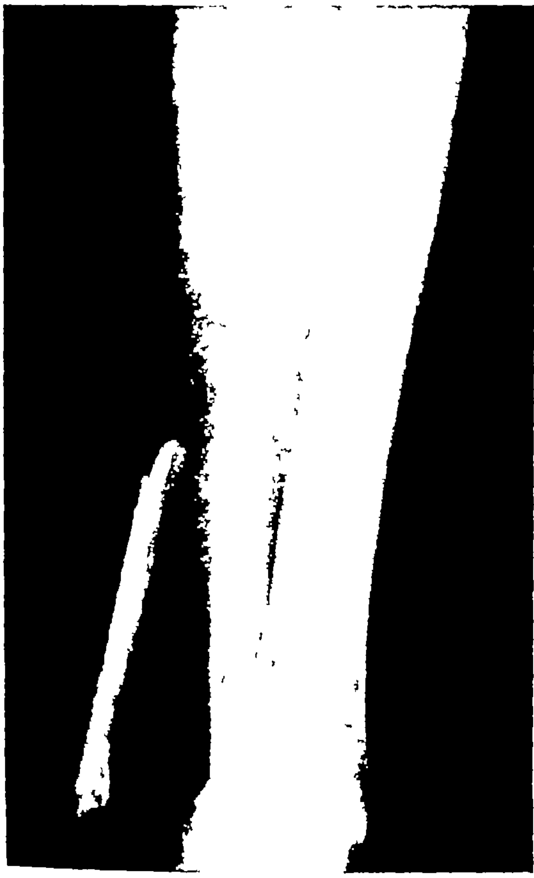


Fig 207

Fig 208

Fig 207 —Ossification of tendo achillis
Fig 208 —Extensive fibrosis of tendo achillis

Fig 209 —Ossification of insertion of tendo achillis

Asymptomatic cases need not be treated If pain ensues and motion is greatly limited, then the entire mass should be excised When the entire tendo achillis must be removed, the terminal end of the triceps surae can be sutured to the peroneus longus and the terminal end of the peroneus longus tendon transferred into the insertion of the tendo achillis

Fig 210 —Osteochondroma at insertion of tendo achillis

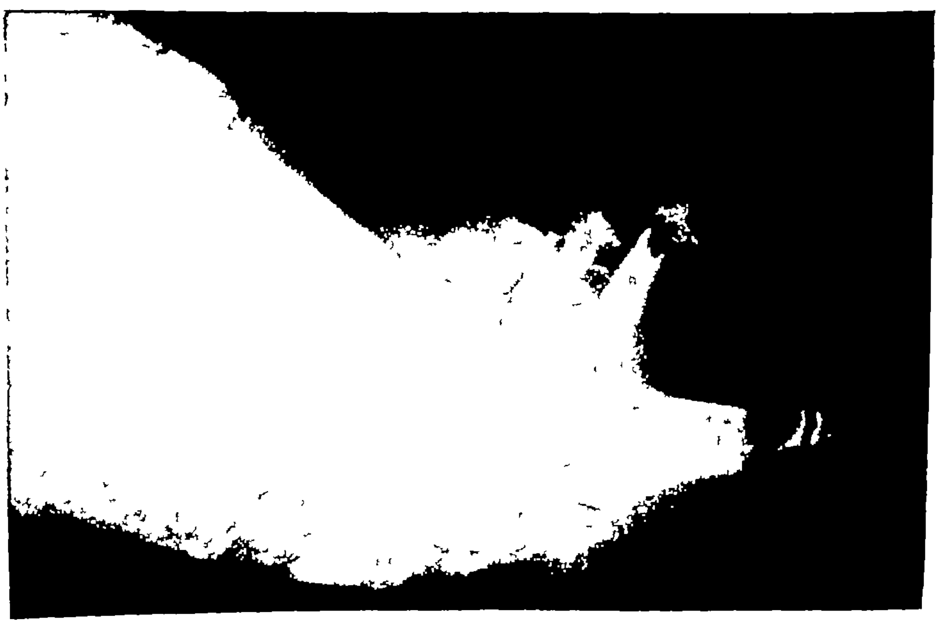


Fig 211 —Ossification of flexor hallucis brevis tendon

OSSIFICATION OF TENDONS OTHER THAN TENDO ACHILLIS

Any tendon in the foot may undergo ossification but seldom does The rare case does not become symptomatic except when the site is under a weight-bearing area or the tendon glides around an angle; then weight bearing or movement of the part becomes painful The diagnosis is made by pinpointing the painful area and confirming ossification of the tendon at that location in the roentgenogram At times, however, ossification may be overshadowed by adjacent bone In a personal case of ossification of the fibular half of the flexor hallucis brevis tendon, pain in the first metatarsophalangeal joint had been constant for three years The patient had to walk on the outer border of the foot The pathologic condition was not disclosed in repeated roentgenograms until one of a series of medioplantar views showed the flexor hallucis brevis tendon ossified from the fibular sesamoid to its insertion (Fig 211) Excision of the fibular sesamoid and the ossified area effected symptomatic cure.

PRINCIPLES OF FREEING A TENDON FROM SCAR TISSUE

The two hindrances to the success of surgical correction of tendons are nonunion and immobilization of the tendon in the scar

Asepsis is imperative. Surgical trauma must be held to an absolute minimum All factors or substances that may induce further scarring are to be scrupulously avoided, tissue reaction to surgical trauma may produce as much scarring and binding of a tendon as infection The structure of the tendon and its surrounding tissues is delicate and must be handled considerately

A tendon cannot glide through a scar, therefore, all scar tissue must be excised Scar tissue left in place heals with additional scarring and again binds the tendon Surrounding skin should be underscored from the deep fascia, to allow free motion with the rest of the skin The tendon should be provided with a new bed of healthy tissue, preferably of fat tissue, especially paratenon, which may be obtained from other tendons The fat should then be covered with a piece of fascia which can be obtained from fascia lata Motion should begin early and slowly and increased gradually in frequency and degree If a tendon is freed from the scar until it moves freely but is then permitted to rest in its bed for a long period, the tendon will adhere to a new scar

Tenotomy—Sectioning of a tendon is performed for contractions in areas where a muscle can be spared This is often done for dorsal contraction of the lesser toes

Sectioning of Tendon—One of the branches of the extensor digitorum longus is severed over the metatarsophalangeal joint The capsule over this joint must usually also be sectioned, because it is often equally concerned in the deformity

1 Make an incision on either border of the tendon
2 Retract the skin margins
3 Press the tenotomy blade against the tendon and metatarsophalangeal capsule and section transversely
4 Close the skin as usual

5 Overcorrect the toe plantarwise and hold in this position for five weeks This allows the tendon and capsule to heal in lengthened position

Tenotomy Through a Stab Wound —The procedure is as follows

1 Place a tenotomy knife on either side of the tendon and force the point of the knife either under or over the tendon

2 Turn the sharp edge against the tendon and force it up or down, depending on the original position of the blade

3 Take care not to sever the skin margin transversely which produces a T-shaped incision It is unnecessary to suture the skin unless a T-shaped incision is made by accident, in which case the transverse part of the incision must be sutured, otherwise an annoying triangular scar will remain

4 Immobilize the toe in a plantar-flexed position for five weeks

Tendon Transference.—Transference of a tendon is often wrongly referred to as *tendon transplantation,* which implies an excision of all or part of a tendon, transferred as a free graft The procedure intended here is the transference of the insertion of a tendon from its original location so as to assume the function of a paralyzed muscle The transference may be into the tendon of another muscle or into bone for the purpose of strengthening a given motion The surgeon must be certain that the tendon to be transferred reaches the area into which it is to be implanted, and even more importantly, he must be certain that the donor tendon can be spared, otherwise a new deformity will result (Figs 212 and 213)

1 Expose the donor tendon at its insertion and at its musculotendinous origin, and detach it at the most distal portion of the insertion

2 Make a second incision over the recipient area, exposing the tendon or bone that is to receive the donor tendon

3 Underscore a new track for the donor tendon to follow from the proximal end of the donor tendon to the insertion of the recipient tendon The new tunnel must pass under any ligaments along its course

4 Thread the donor tendon through the new tunnel by means of long forceps or a Bunnell tendon guide, making certain that the donor tendon has no kinks or twists along its new course which may cause atrophy of the muscle

5 Place the foot in an overcorrected position to permit the tendons to remain in a relaxed position which is held until final immobilization

6 Braid sutures into both tendons and the periosteum of the bone If a larger tendon is transplanted into a smaller tendon, the smaller is slit, the larger passed through this opening, and the end sutured below the slit Suture the skin in the usual manner

7 Immobilize the foot in a cast or splint in an overcorrected position for six to eight weeks

Bunnell's stainless steel-button anchoring technique, which conveniently modifies standard procedure, is probably more satisfactory for tendon transference in the foot (Fig 355)

1 Make an incision over the terminal end of the donor tendon, freeing it from its insertion

2 Make a second incision at the musculotendinous junction of the donor muscle and withdraw the entire tendon through that incision

3 Make a third incision over the recipient area

4 Make a new tunnel, beginning from the second incision and running to the third incision, making certain that the tunnel passes under all important ligaments along its course

5 With two long straight needles, thread stainless steel wire through the donor tendon, beginning at its proximal end and having it escape at its distal end

6 Insert a pull-out wire under the proximal loop of the threaded wire

7 Guide the two straight needles through the tunnel and follow with the donor tendon

Fig 212

Fig 213

Fig 212 —Plantar contraction of lesser toes, resulting from transference of extensor longus digitorum

Fig 213 —Plantar contraction of all toes, resulting from transference of extensor longus digitorum and extensor longus hallucis

8 In most cases of tendon transference in the foot, it is preferable to insert the tendon into bone If the tendon is to be inserted into bone, drill through the recipient bone a hole large enough for the donor tendon to pass through

9 Pass both straight needles through the bone aperture and through the skin under the aperture, carrying the distal end of the tendon into the bone aperture.

10 Pass the needles through a button and tie the wires over the center bar of the button while holding the foot in an overcorrected position

11 Pack the bone chips from the drilling around the tendon in the bone aperture

12 Close the three incisions, allowing the pull-out wire to escape through the first incision and lie over gauze on the skin.

13 Apply a plaster cast from the toes to above the knee with the foot in an overcorrected position so as to avoid all strain on the transferred tendon

14 Bivalve the cast after twenty-four hours, and keep the limb suspended and at complete rest for ten days, after which remove the cast shell, dress the wound, and remove any irritating sutures other than the stainless steel wire Apply a cast and walking heel, up to the knee

15 Remove the cast eight weeks later, cut the wires over the button bar, and by means of the pull-out wire remove the wire in the tendon

16 Mild motion and light weight bearing may now begin

17 Normal physiologic action of the transferred muscle must be established by slow retraining on the part of the patient

Tendon Lengthening —Lengthening of a contracted tendon is necessary when the muscle cannot be spared The extensor hallucis longus is the one commonly contracted tendon of the foot that lends itself to lengthening The contracture is usually accompanied by hallux valgus If it is not lengthened at the time of reduction of hallux valgus, hallux flexus occasionally takes place even though the reduction was done properly

1 Make an incision about 3 to 6 cm long on either side of the tendon

2 Incise the tendon sheath longitudinally to the same extent as the skin incision

3 Incise the exposed tendon transversely half its width on one side at the proximal end of the longitudinal incision and half its width on the other side at the distal end of the incision, the distance between the two transverse incisions should be about the amount of lengthening desired

4 Join the two transverse incisions by a longitudinal incision

5 Stretch the part into the desired position The ends may or may not require suturing

6 Replace the sheath and paratenon over the tendon, and suture the skin Immobilize the foot in a cast or splint in the overcorrected position for three to five weeks

Gueukdjian (1956) has suggested an ingenious method of transferring a small portion of the distal part of the long saphenous vein as a cuff over the lengthened or repaired part of a small tendon, such as the extensor hallucis longus This assures a good gliding mechanism

SHORT TENDO ACHILLIS

Lengthening of the tendo achillis is a safe and useful procedure in selected cases in which dorsiflexion of the foot is limited more than 105 degrees. The procedure is often a minor part in the surgical correction of talipes when major alteration of tarsal articulations are undertaken In many cases of acquired or congenital talipes equinus, this simple procedure suffices.

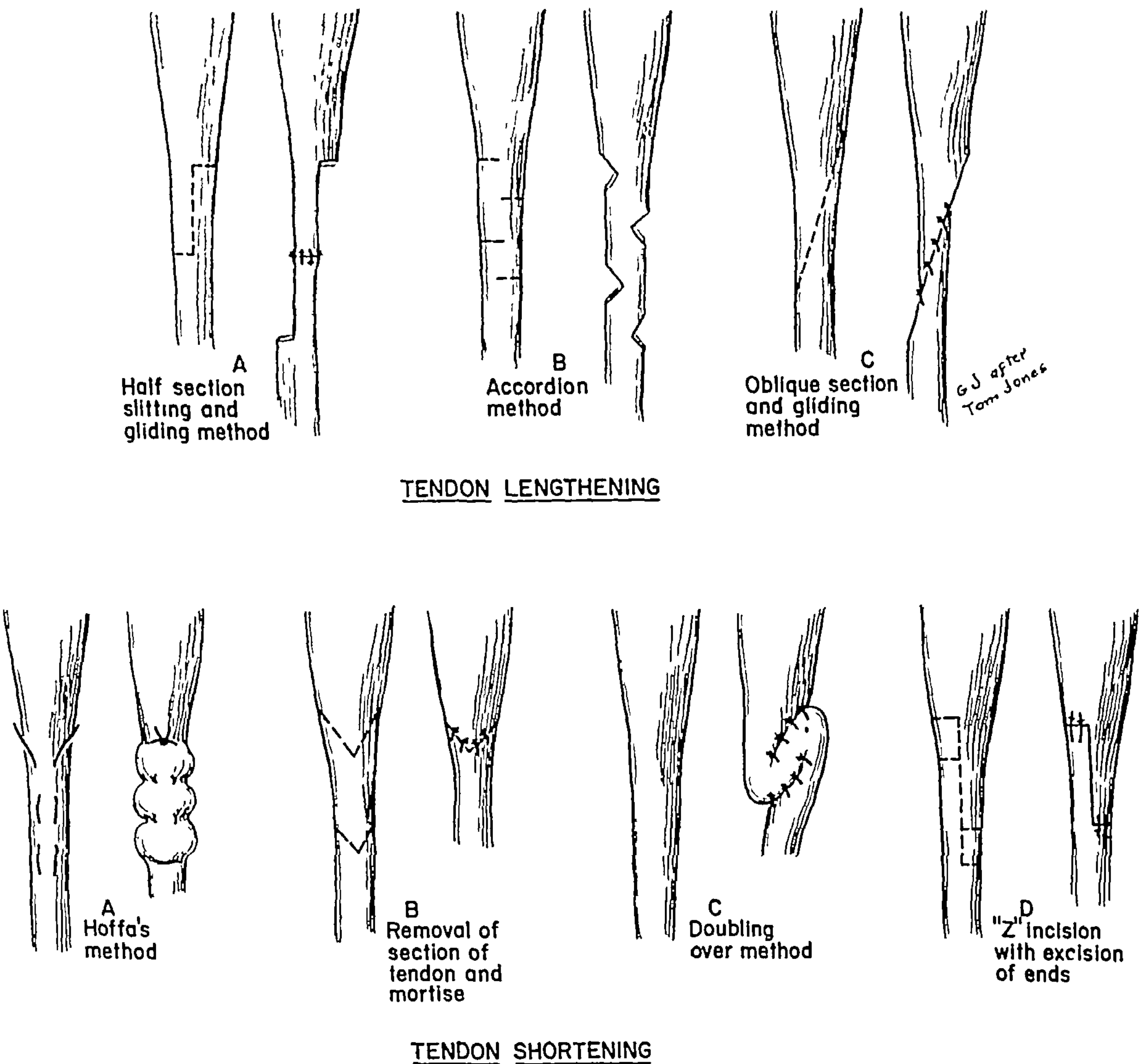


Fig 214 –Different procedures for tendon lengthening and tendon shortening

Many techniques have been devised to lengthen the tendo achillis, from the simple subcutaneous tenotomy to Vulpius' procedure for lengthening of the fascia over the gastrocnemius (Fig 214) Hodgen and Frantz (1938) studied the relative merits of plastic operations and subcutaneous tenotomy for lengthening the tendo achillis They questioned twenty-five orthopedic surgeons, twenty-one opposed subcutaneous procedure or questioned its worth, because in some cases the tension of the triceps surae produces separation of the tendon, function of the calf muscles is then taken over by the peroneus longus and tibialis posticus,

which often results in a pes cavus The procedure of choice and the one most widely practiced is the Z-plasty operation in the anteroposterior or mediolateral plane

Recommended Z-Plasty Technique Mediolateral Plane.—This procedure has an advantage in that the exposed raw surface of the tendon is proximal, therefore relatively deep, and can be covered with subcutaneous tissue and fat, whereas the cut surface of the lower part of the tendo achillis is deep, preventing its becoming adherent to the skin

1 Make an incision about 8 to 10 cm long on the medial side of the tendo achillis, incise the tendon sheath and retract its edges

2 Incise the tendon from side to side longitudinally for a distance equal to the desired increase in length, or about 4 to 7 cm

3 Sever the posterior part of the divided tendon at its proximal end and the anterior part at its distal end

4 At this stage the foot usually can be fully dorsiflexed and the cut opposed edges sutured without tension

The procedure for lengthening the calcaneus tendon in the anteroposterior plane is the same

Capsulotomy of Posterior Tibiotalar Joint.—If the deformity cannot be reduced following lengthening of the tendon, a capsulotomy of the posterior tibiotalar joint, as described by Brockman (1930), is indicated The tendons, nerves, and vessels behind the medial malleolus are gently freed and retracted, exposing the tibiotalar capsule which can be incised transversely, thereby permitting dorsiflexion Capsulotomy, as called attention to by Brockman, may be employed in addition to any type of lengthening of a heel cord

Before the leg is immobilized, it is important to make certain that the skin over the heel cord is not under excessive tension, because too much stretching of the skin may cause it to slough and the resultant scar may produce a worse deformity If the skin is taut, the foot may be only partly dorsiflexed and should be maintained in a splint for a week or two, when further correction can be obtained without danger of sloughing A cast from the toes to mid-thigh is applied with the knee in semiflexion The cast is worn for about six weeks, followed by passive and active exercise Walking with a drop-foot brace is then begun The brace should be worn for about six months

REPAIR OF DEFECTS IN TENDO ACHILLIS

Repairing an old ruptured tendo achillis can be a formidable problem Transference of the peroneus longus or tibialis posticus tendon into the stump of the tendo achillis has been suggested Bosworth (1956) described an autogenous tendon graft performed successfully in six cases of old defects in the heel cord He excised a longitudinal pedicle graft about 1 cm wide from the entire length of the proximal portion of the ruptured tendon The distal end of the graft is not excised but is left in place as a pedicle The free tongue of the graft is next woven into both stumps of the ruptured tendon and sutured in this position (Fig 215) This is followed by application of a mid-thigh cast, with the foot in plantar flexion Weight bearing is not permitted for six weeks, at which

time the cast is removed During the next six weeks, weight bearing is gradually resumed

TENDON LUXATION

Tendon luxation is a slipping of a tendon from its natural groove It is usually associated with congenital malformation and deformities, however, it may result from injury, arthritis, or malunited fractures The most symptomatic tendon luxation of the foot is that of the peroneus longus

Luxation of the peroneus longus tendon is due to a poorly formed groove on the posterior surface of the lateral malleolus which permits the tendon of the peroneus longus to slip over it It is characterized by recurrent episodes of sudden slipping of the peroneus longus tendon while walking This slipping often makes the patient fall, because the peroneus longus goes immediately into spasm, thereby abducting and plantar-flexing the foot The condition can usually be reduced manually But the attacks tend to recur with increasing frequency, because the lateral fibulocalcaneal ligament is stretched farther with each episode.

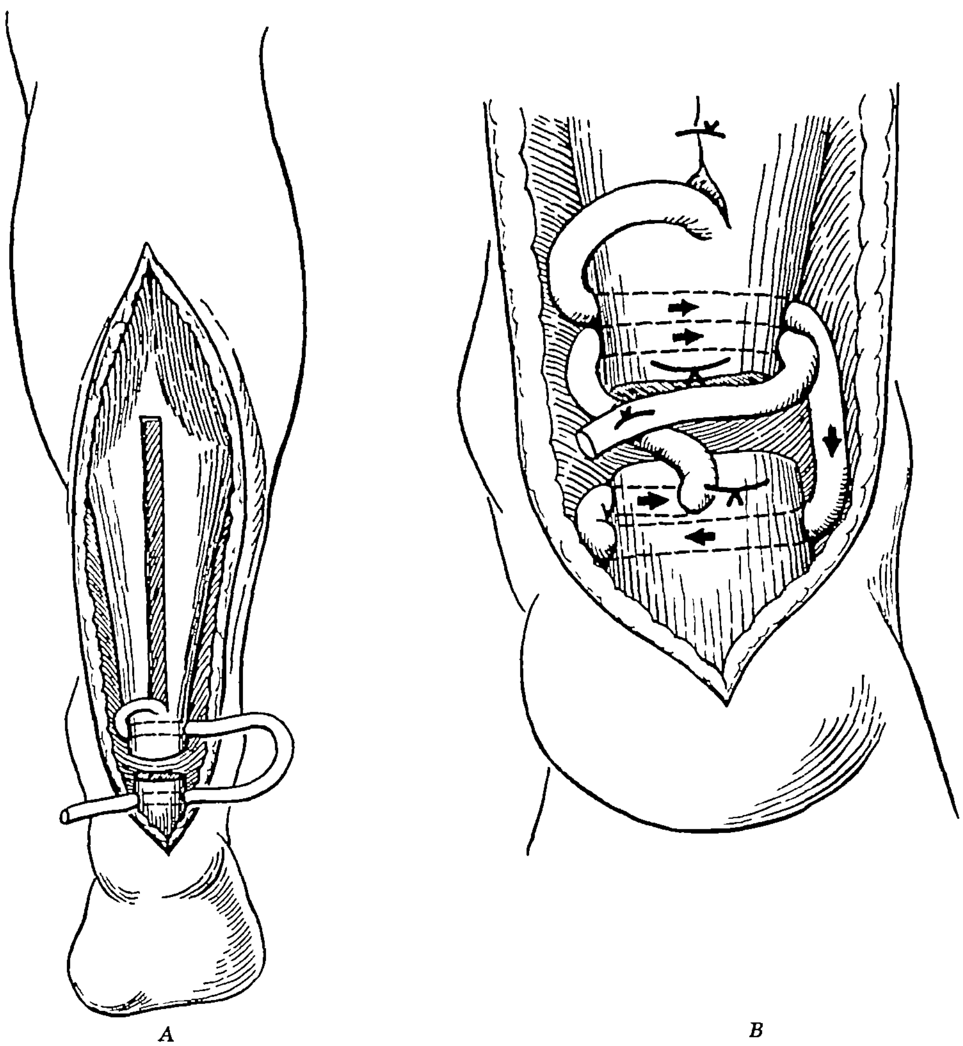


Fig 215 —Bosworth's repair of tendo achillis *A*, Vertical graft excised from tendo achillis, from above downward, leaving it attached at distal end *B*, Graft threaded through proximal and distal ends of tendo achillis and sutured to itself Defect left by graft is sutured

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in the posterior aspect of the malleolus is shallow or if the groove in the lower end of the fibula is shallow, the Kelly technique should be followed. After the field has been exposed as described under the recommended technique for correction, a transverse wedge-shaped section about 2 cm wide is cut out of half of the depth of the lateral malleolus The wedge is pushed posteriorly for about 0 5 cm, so that its tip hangs over the peroneal tendons The wedge is fixed in this position with an autogenous bone peg or small screw (DuVries' modification, Fig 216) The ligaments and fascia are closed with chromic gut, and the skin is closed as usual.

Modification of Kelly Procedure by Watson-Jones (1955) Watson-Jones suggests a simplification of Kelly's procedure He forms a thick osteoperiosteal flap cut from the surface of the lateral malleolus, with an intact pedicle of periosteum and soft tissue, which is swung back and held in position by one or two catgut sutures

Postoperative Care.—A circular walking cast to the knee is worn for eight weeks Following removal of the cast, gradual walking is permitted Normal function may be expected in about two months.

REFERENCES

Bosworth, D M Repair of Defects in the Tendo Achillis, J Bone & Joint Surg **38A** 111, Jan , 1956

Brockman, E P Congenital Club-Foot, London, 1930, John Wright & Sons, Ltd

Bunnell, Sterling Surgery of the Hand, ed 3, Philadelphia, 1956, J B Lippincott Co

Burman, M Stenosing Tendovaginitis of Foot and Ankle, A M A Arch Surg **67** 686, Nov , 1953

Burman, M Subcutaneous Tear of the Tendon of the Peroneus Longus, A M A Arch Surg **73** 216, Aug , 1956

Burman, M Subcutaneous Rupture of the Tendon of the Tibialis Anticus, Ann Surg **100** 368, Aug , 1934 (Cited by Mensor and Ordway)

Christie, B G B Diagnosis and Treatment of Tenosynovitis, Brit M J **1** 1501-1503, June 25, 1955

Cohen, H , and Reid, J B Tenosynovitis Crepitans Associated With Oxaluria, Liverpool Med Chir J **43** 193, part 3, 1935 (Cited by Lapidus, 1950)

Els, H Ueber eine Abrissfraktur des Tibialis-anticus-Ansatzes, Deutsche Ztschr f Chir **106** 610, Sept., 1910 (Cited by Burman, 1934)

Ghormley, J W Ossification of the Tendo Achillis, J Bone & Joint Surg **22** 153, Jan , 1938

Ghormley, J W , and Spear, I M Anomalies of Posterior Tibial Tendon, Cause of Persistent Pain About Ankle, A M A Arch Surg **66** 512, April, 1953

Gueukdjian, S A A New Method of Canalizing Tendon Sutures With Vein Grafts, A M A Arch Surg **73** 1018, Dec , 1956

Hodgen, J T , and Frantz, C H Subcutaneous Tenotomy of Achilles Tendon, J Bone & Joint Surg **20** 419, April, 1938

Horing, F Ueber Tendinitis ossificans traumatica, Munchen med Wchnschr **55** 674, March, 1908

Jacobsthal, H Ueber Fersenschmerzen ein Beitrag zur Pathologie des Calcaneums und der Achillessehne, Arch klin Chir **88** 146, part I, 1908-1909

Kelly, R E An Operation for Chronic Dislocation of Peroneal Tendons, Brit J Surg **7** 502, 1920

Lapidus, P W Indirect Subcutaneous Rupture of Anterior Tibial Tendon, Report of Two Cases, Bull Hosp Joint Dis **2** 119, July, 1941

Lapidus, P W , and Seidenstein, H Chronic Non-Specific Tenosynovitis With Effusion About the Ankle, J Bone & Joint Surg **32A** 175, Jan , 1950

Lipscomb, P R , and Kelly, P J Injuries of Extensor Tendons in Distal Part of Leg and in Ankle, J Bone & Joint Surg **37A** 1206, Dec , 1955

Mensor, M C , and Ordway, G L Traumatic Subcutaneous Rupture of Tibialis Anterior Tendon, J Bone & Joint Surg **35A** 675, July, 1953

Watson-Jones, Reginald Fractures and Joint Injuries, ed 4, Baltimore, 1955, Williams & Wilkins Co , p 830

Treatment.—At first occurrence, the deformity is reduced and the foot immobilized for five to eight weeks in a plaster cast There are often no recurrences If there are recurrences, surgical intervention is indicated

Recommended Technique to Correct Tendon Luxation of the Peroneus Longus—If the peroneal groove is of normal depth, it can be assumed that the luxation was caused by avulsion or stretching of the fibulocalcaneal ligament and that repair and reinforcement will suffice

1 Make an incision immediately behind the lateral malleolus, beginning about 6 cm. above the tip of the malleolus, extending it distally and rounding it under the tip to a point over the lateral surface of the cuboid

2 Retract the skin margins and make a similar incision in the fibulocalcaneal ligament and fascia (peroneal retinaculum) which encase the peroneal tendons

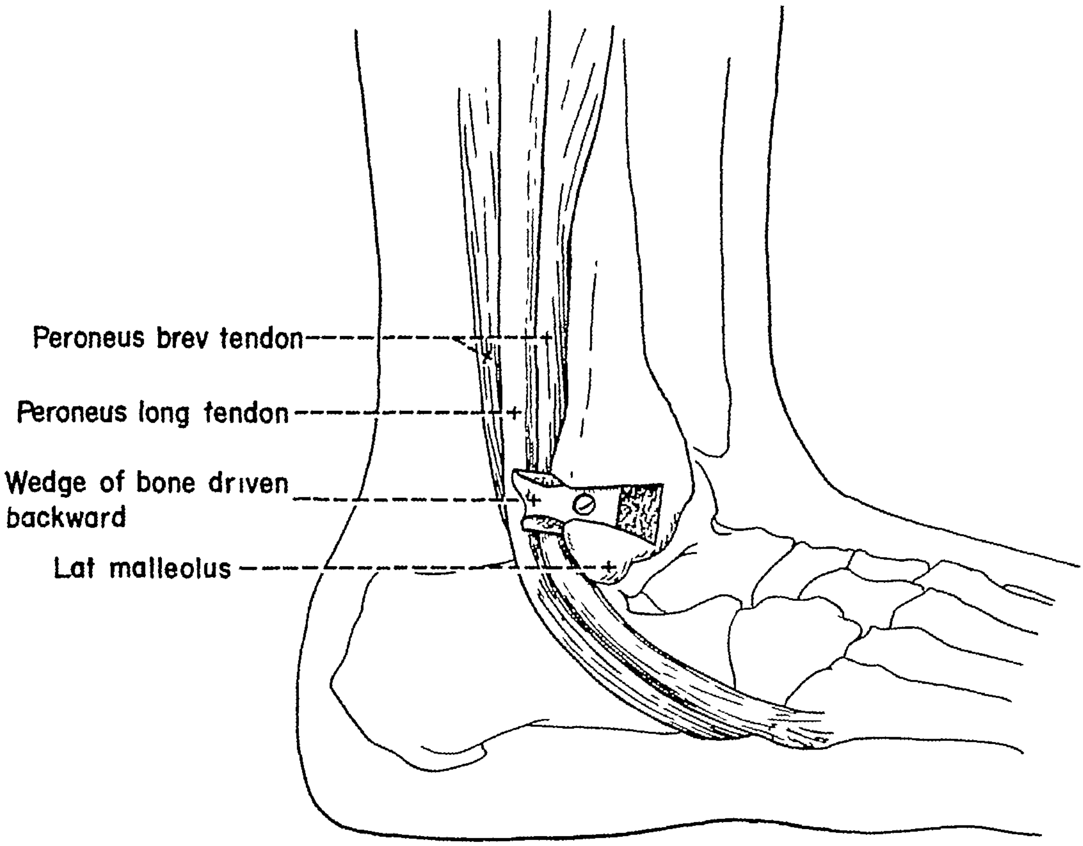


Fig 216—DuVries' modification of Kelly's wedge bone graft for peroneus longus subluxation Wedge osteotomy made in lateral malleolus, graft pushed backward and stabilized with small screw

3. Retract the fascial margins to expose the peroneal tendons and the peroneal groove of the malleolus

4 Suturing the incised ligament usually removes any slack that may have been present, the scar that forms in the suture line further reinforces the ligamentous structure A piece of fascia lata 2 by 4 cm grafted over the ligament suture line gives still further reinforcement

Kelly Technique, Recommended Application—When the peroneal groove is shallow or malformed, Kelly's operation is a successful procedure. If the groove

SIMPLE SESAMOIDITIS

Simple sesamoiditis is an inflammation and swelling of the peritendinous structures of the sesamoids The tibial sesamoid is involved most, because it bears most of the weight carried by the first metatarsal head The condition may be a symptom in a syndrome of a disease, such as a neuropathy due to syphilis or diabetes; or it may be of local origin, as a result of injury or infection.

The commonest type is due to a weak foot, jumping, excessive dancing, wearing short shoes or high-heeled shoes, or to the presence of an abnormally large

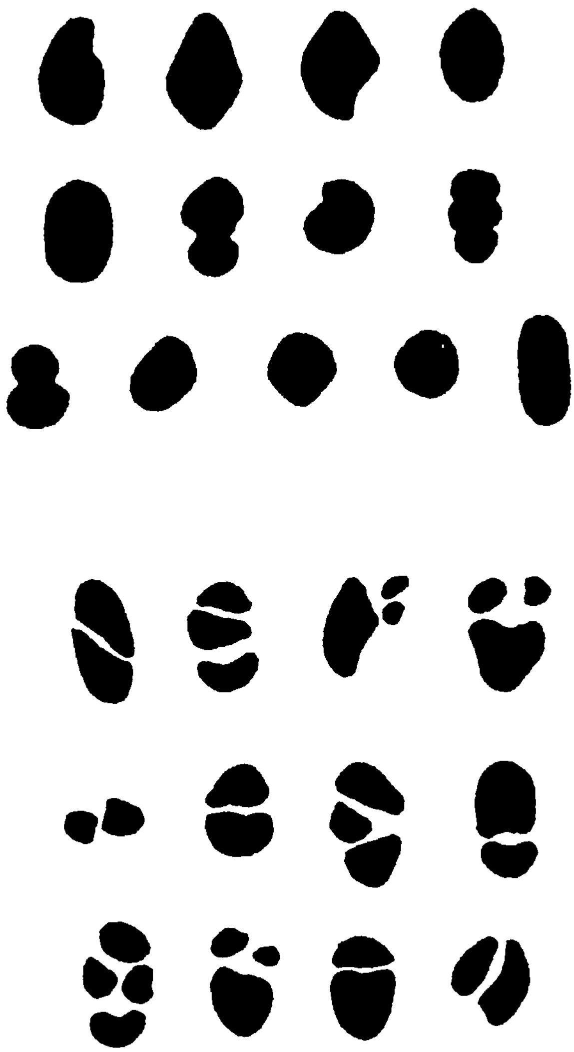


Fig 217 —Some variations in size and shape of hallux sesamoids

or an anomalously shaped sesamoid The type of sesamoid likely to produce symptoms is that located forward where it nearly touches the joint space (Fig 219) The disorder may be acute or chronic, depending on the causative factors

Symptoms.—The onset may be sudden or gradual, with pain on weight bearing and on dorsiflexion of the great toe Sesamoids are tender on palpation, often accompanied by edema and swelling under and around the first metatarsal head In chronic or recurrent cases, a large adventitious bursa may develop (page 228)

CHAPTER TWELVE

Disorders of the Sesamoids

THE TERM SESAMOID IS SAID TO HAVE BEEN FIRST APPLIED BY GALEN AND IS TAKEN from the small bone's resemblance to the sesame seed (Inge and Ferguson, 1933) The only two areas that normally contain such bones are the patella over the knee joint and the head of the first metatarsal, under which are two sesamoids With varying frequency, however, sesamoids do develop in many tendons that glide over joints, mostly in the foot, but those are ordinarily only of academic interest The hallux sesamoids are always present They are of practical interest as a common cause of foot disabilities Their wide variation in size and shape (Fig 217) is one reason for their troublesomeness The occasional accessory sesamoid of the foot may also produce disabling symptoms

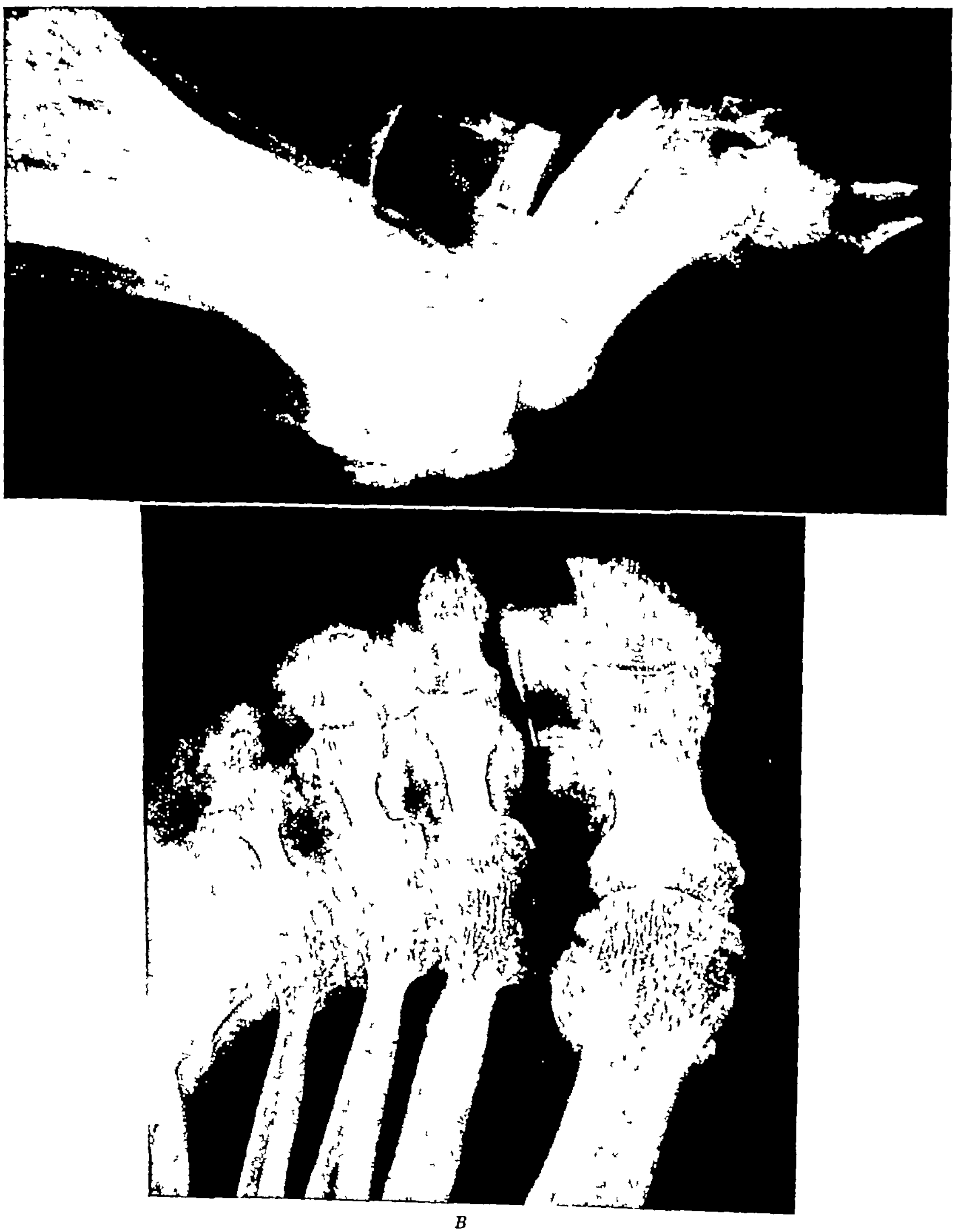
Sesamoids under the great toe joint are an essential part of the first metatarsophalangeal joint ensemble and are incorporated in the flexor hallucis brevis tendon The great toe joint is by far the most important functioning structure of the forefoot The ossification of the hallux sesamoids takes place between the seventh and tenth year, often arising from multiple centers and resulting in bipartite and tripartite types of sesamoids Sesamoids are generally thought to be inconsequential and receive scant attention except by writers on hallux valgus, who point out the influence of sesamoids in the causation of this serious deformity (Toepel, 1929, Inge, 1936, Gottlieb, 1941) Kewenter (1936) studied 800 roentgenograms of adult feet and 465 of children's feet He reported an incidence of 35 5 per cent divided sesamoids, mostly in the tibial sesamoid

Accessory sesamoids may occur under any or all of the metatarsal heads (Fig 218), under the head of the first proximal phalanx, and in the tendon of the foot, especially where it glides over such a bone as the peroneus longus The accessory sesamoids only at times become symptomatic

DEGENERATION AND ASEPTIC NECROSIS OF SESAMOIDS

Degeneration of the sesamoids may involve one or both sesamoids as a result of infection or degenerative diseases The condition is rare The suppurative type is usually due to a pyogenic organism which invades the area throughout an abrasion Adjacent bone structures may or may not be infected The non-

A



B

Fig 220 —A, Medial view Aseptic necrosis of tibial sesamoid secondary to diabetes mellitus B, Dorsiplantar view showing also degenerative changes of fibular sesamoid

Treatment.—In early stages, complete rest or removal of weight bearing on the sesamoid by means of padding or appliances often resolves the problem In recurrent or intractable cases, excision of the sesamoid is indicated (page 194)

Fig 218 —Accessory sesamoids under all lesser metatarsals

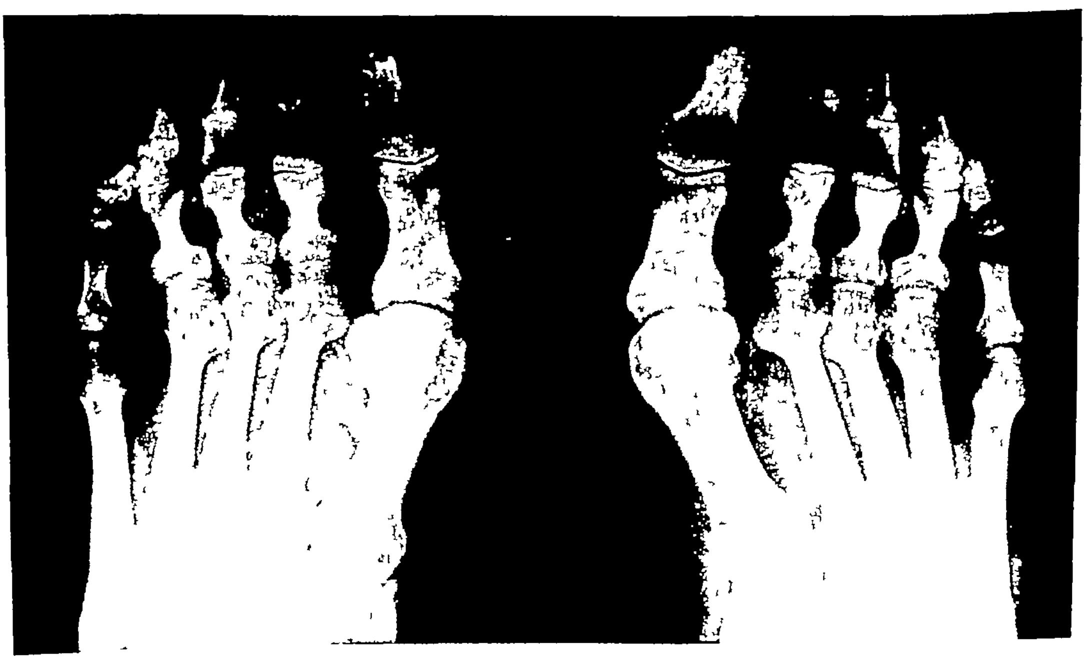


Fig 219 —Sesamoids articulating near first metatarsophalangeal joint space likely to become symptomatic

A

B

Fig 222 —A, Dorsiplantar view of dislocation of distal half of bipartite sesamoid into first metatarsophalangeal joint B, Oblique views C, Dorsiplantar view of a comparable case

(Fig 222 continued on next page)

suppurative type is usually due to a nonpyogenic organism or to a debilitating disease (Fig 220) or to injury of the peripheral nerve Aseptic necrosis of the sesamoids is occasionally encountered It may result from loss of the blood supply to the sesamoids

Symptoms.—Acute infectious osteomyelitis of the sesamoids has a typical pyogenic course pain, swelling, rise in body temperature, and leukocytosis evidenced soon after onset As soon as organization takes place, accumulation of pus causes fluctuation Pyogenic infection may destroy the sesamoid completely The course of nonsuppurative osteomyelitis of the sesamoids is chronic, characterized by chronic swelling and pain under the great toe joint, and, at times, fluctuation A sanguineous material, usually sterile, is obtained on drainage (Fig 221).

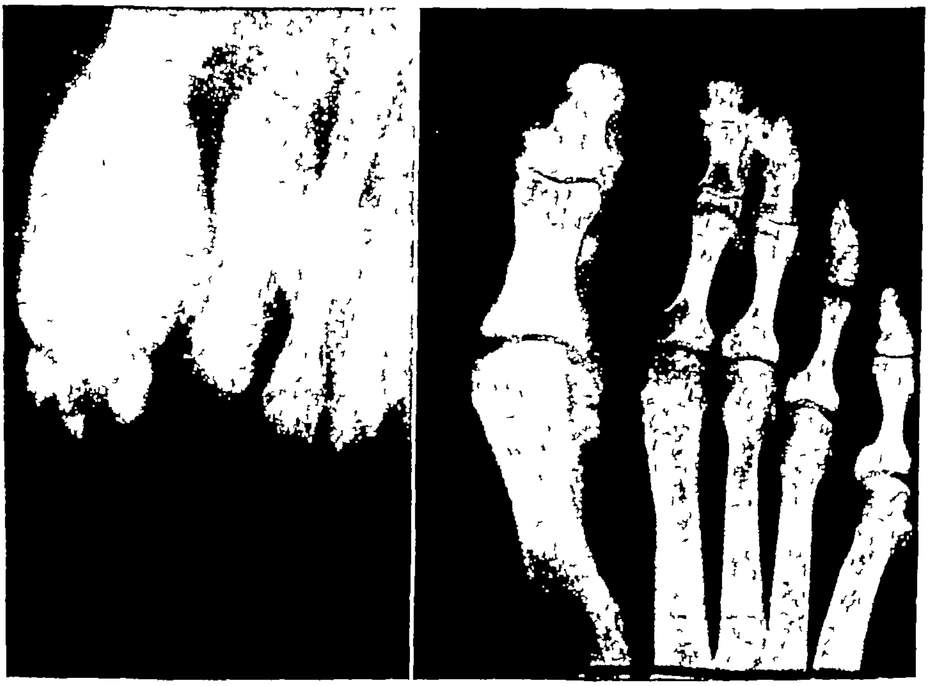


Fig 221 —Nonpyogenic osteomyelitis of both sesamoids, with complete disorganization Process local in origin, of five months' duration, sanguineous material obtained

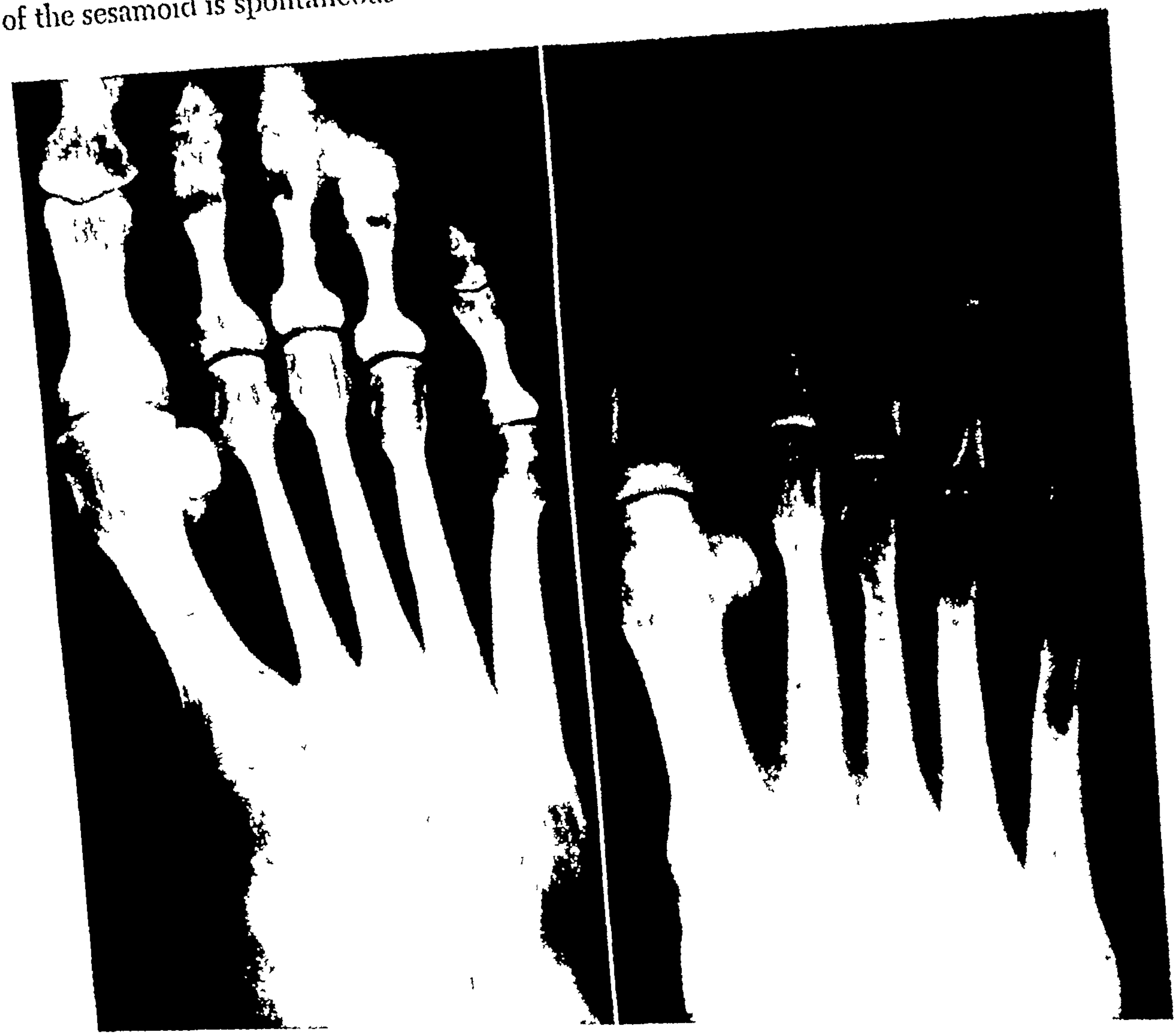
Treatment —Rest, hot fomentations, antibiotics taken internally, and drainage instituted when organization has taken place comprise the treatment for the suppurative type Nonsuppurative osteomyelitis of the sesamoids should be treated palliatively, by padding and distribution of weight to relieve the weight-bearing stress of the sesamoid Palliative measures should be tried first, and then excision of the sesamoid if response fails If the condition is secondary to a systemic disease, the underlying disease must be treated or controlled In such cases, the diseased sesamoid should be excised only as a last resort and then under the most aseptic conditions

DISLOCATIONS OF SESAMOIDS

The commonest dislocation of the sesamoids is a relative displacement of both sesamoids, generally as a result of adduction of the head of the first meta-

jagged and irregular, whereas in bifurcation, the outline is regular and the division, smooth Fracture of the fibular sesamoid is rare (Fig. 225).

Etiology.—Direct trauma, injury incurred by jumping from heights or by excessive dancing of any type, may cause the fracture. In rare instances, fracture of the sesamoid is spontaneous



A B

Fig 223.—A-C, Types of dislocation of fibular sesamoid into metatarsal interspace, without hallux valgus deformity which produced symptoms

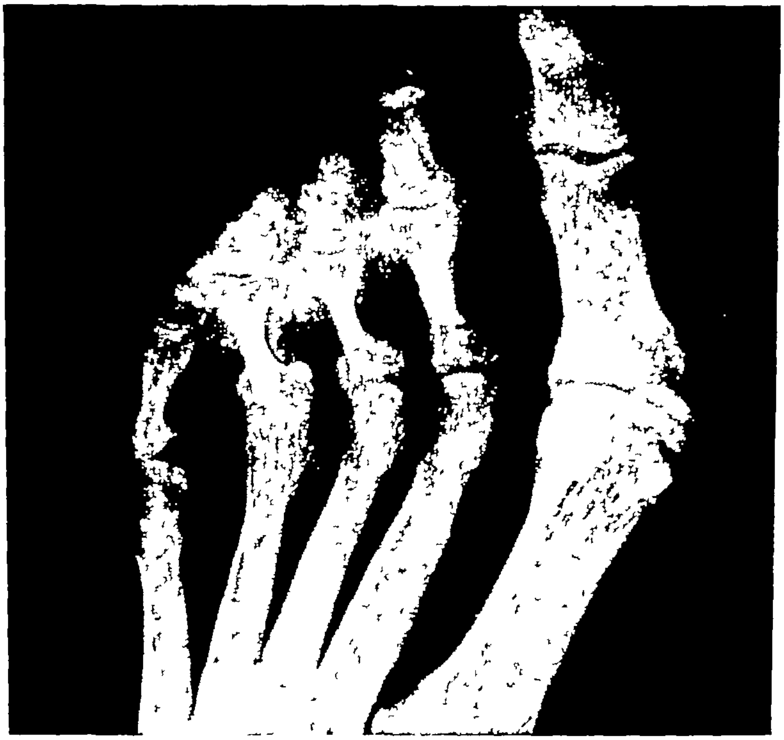
(Fig 223 continued on next page.)

Symptoms.—The onset is sudden On palpation of the sesamoid, especially on dorsiflexion of the great toe, pain is sharp In some cases, pain may be mild at the onset, becoming gradually severe The patient may not seek relief for da or weeks after the accident. The periarticular structures on the plantar of the first metatarsophalangeal joint become swollen.

Treatment.—A recent fracture should be immobilized completely f weeks In many cases palliative measures do not bring response, because layed treatment. Surgical removal of the sesamoid is then inevitable (See nique, pages 194 and 228)

tarsal, as is seen in hallux valgus. This leaves the flexor hallucis brevis and the sesamoids in their original positions, however, each sesamoid may be displaced individually, or a part of a bifurcated sesamoid may be dislocated

Acute dislocation of a sesamoid is likely to involve the tibial sesamoid, rarely is the fibular sesamoid involved The sesamoid is mostly bipartite or tripartite (Fig 222) The dislocation in most cases is of the distal portion of the sesamoid which may become wedged in the metatarsophalangeal joint Pain is sudden and severe Pain is aggravated by dorsiflexion of the great toe Swelling and limitation of motion of the first metatarsophalangeal joint are typical



C

Fig 222 (cont'd) —For legend see page 261

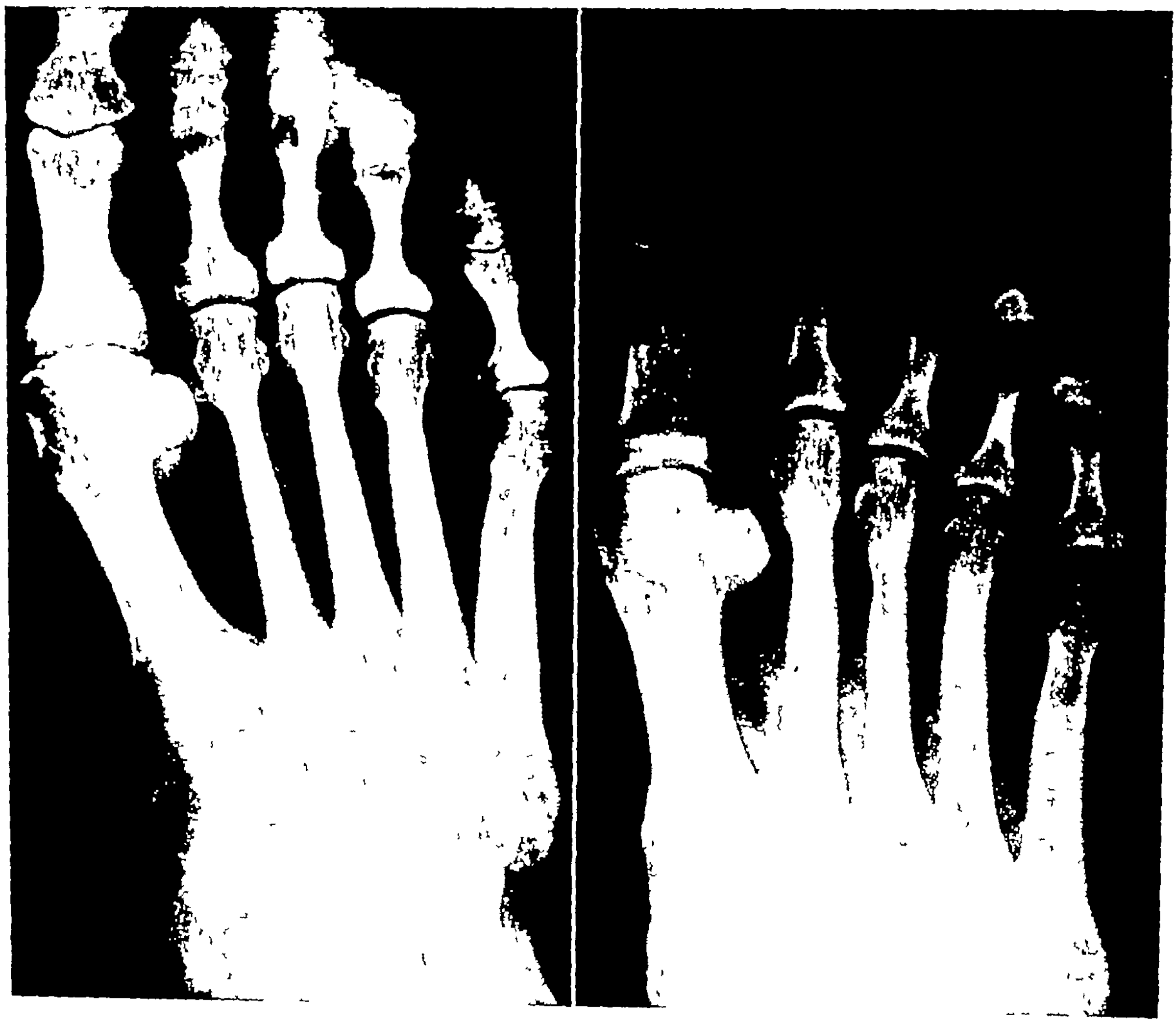
Chronic dislocation of the fibular sesamoid without hallux valgus is sometimes encountered and may become symptomatic (Fig 223) The condition typically requires excision of the entire bone through a dorsal incision of the first metatarsal interspace (See Technique, page 383) Treatment for dislocated tibial sesamoid requires the excision of the entire sesamoid through an incision over the medial plantar border of the great toe joint (See pages 194 and 228)

FRACTURE OF SESAMOIDS

The tibial sesamoid is the one most likely to fracture, because normally it receives most of the weight transmitted by the first metatarsal The fracture is usually transverse or comminuted (Fig 224) The condition must always be differentiated from a normal bifurcation In a true fracture, the line of division is

jagged and irregular, whereas in bifurcation, the outline is regular and the division, smooth Fracture of the fibular sesamoid is rare (Fig 225)

Etiology.—Direct trauma, injury incurred by jumping from heights or by excessive dancing of any type, may cause the fracture In rare instances, fracture of the sesamoid is spontaneous



A B

Fig 223—*A-C*, Types of dislocation of fibular sesamoid into metatarsal interspace, without hallux valgus deformity which produced symptoms

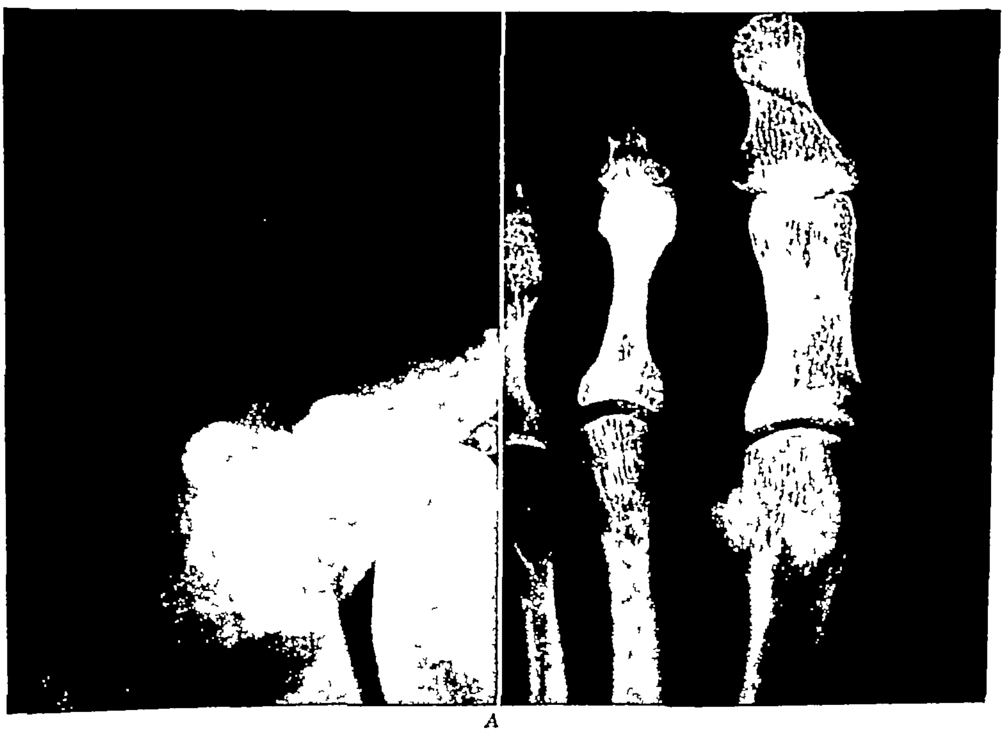
(*Fig 223 continued on next page*)

Symptoms—The onset is sudden On palpation of the sesamoid, especially on dorsiflexion of the great toe, pain is sharp In some cases, pain may be mild at the onset, becoming gradually severe The patient may not seek relief for days or weeks after the accident The periarticular structures on the plantar surface of the first metatarsophalangeal joint become swollen

Treatment—A recent fracture should be immobilized completely for three weeks In many cases palliative measures do not bring response, because of delayed treatment Surgical removal of the sesamoid is then inevitable (See Technique, pages 194 and 228)

C

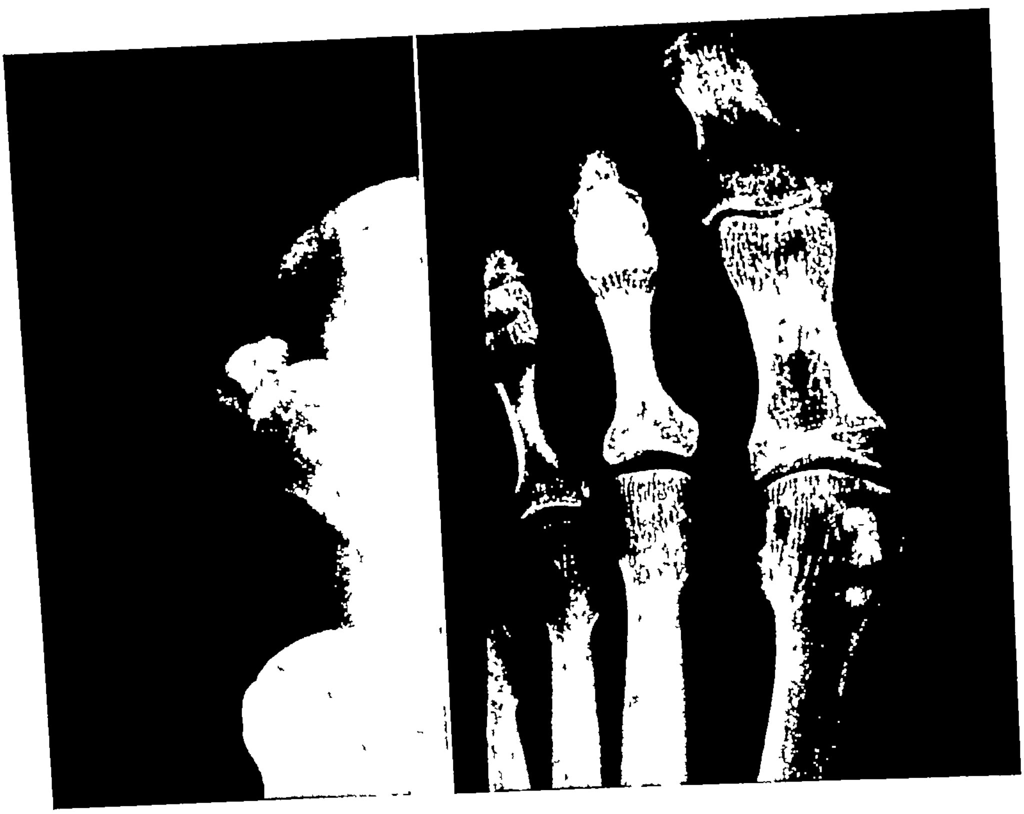
Fig 223 (cont'd) —For legend see page 263



A

Fig 224 —*A*, Transverse fracture of tibial sesamoid *B*, Comminuted fracture of tibial sesamoid *C*, Fracture of proximal portion of tibial sesamoid

B



C

Fig 224 (cont'd) —For legend see opposite page

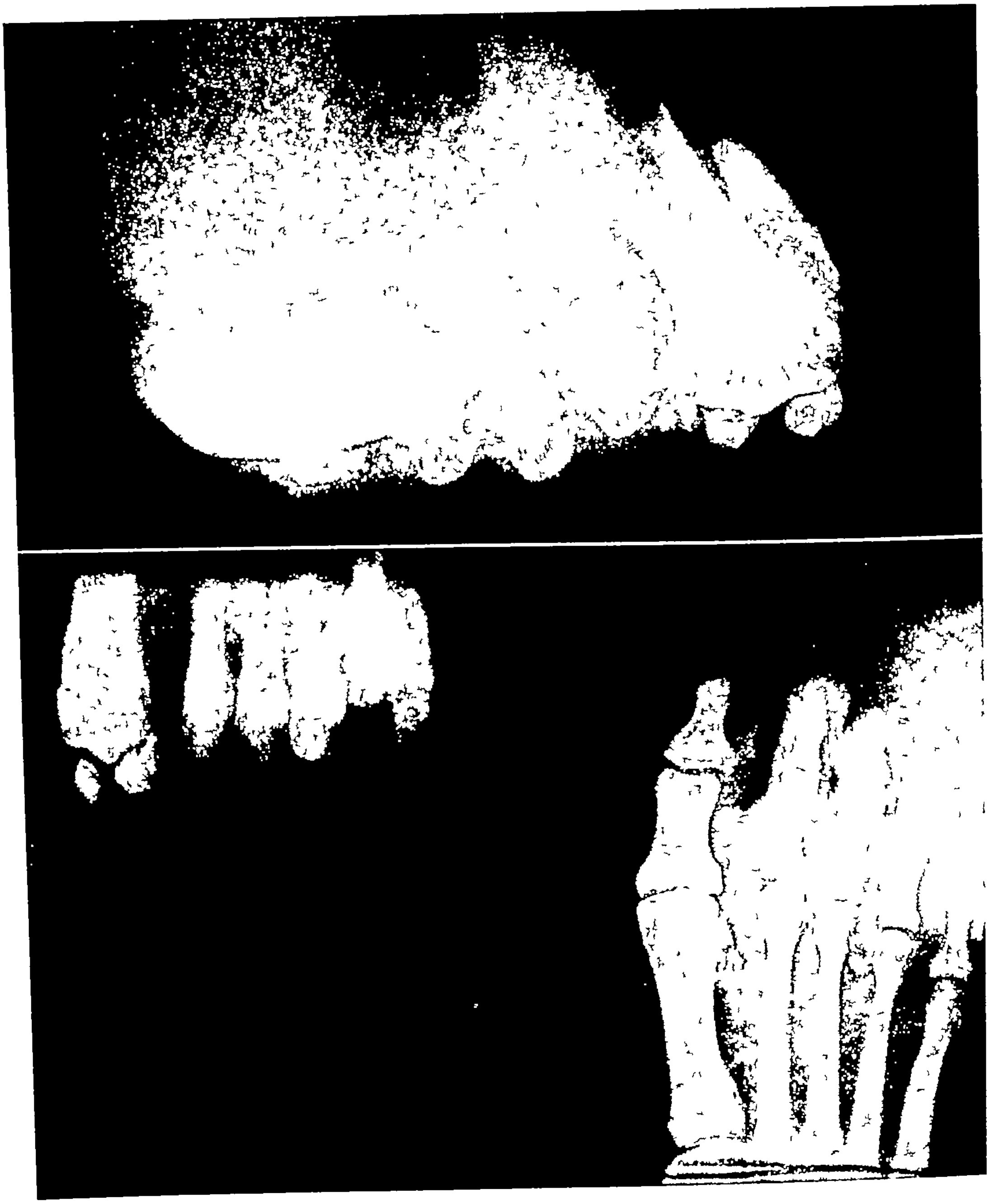


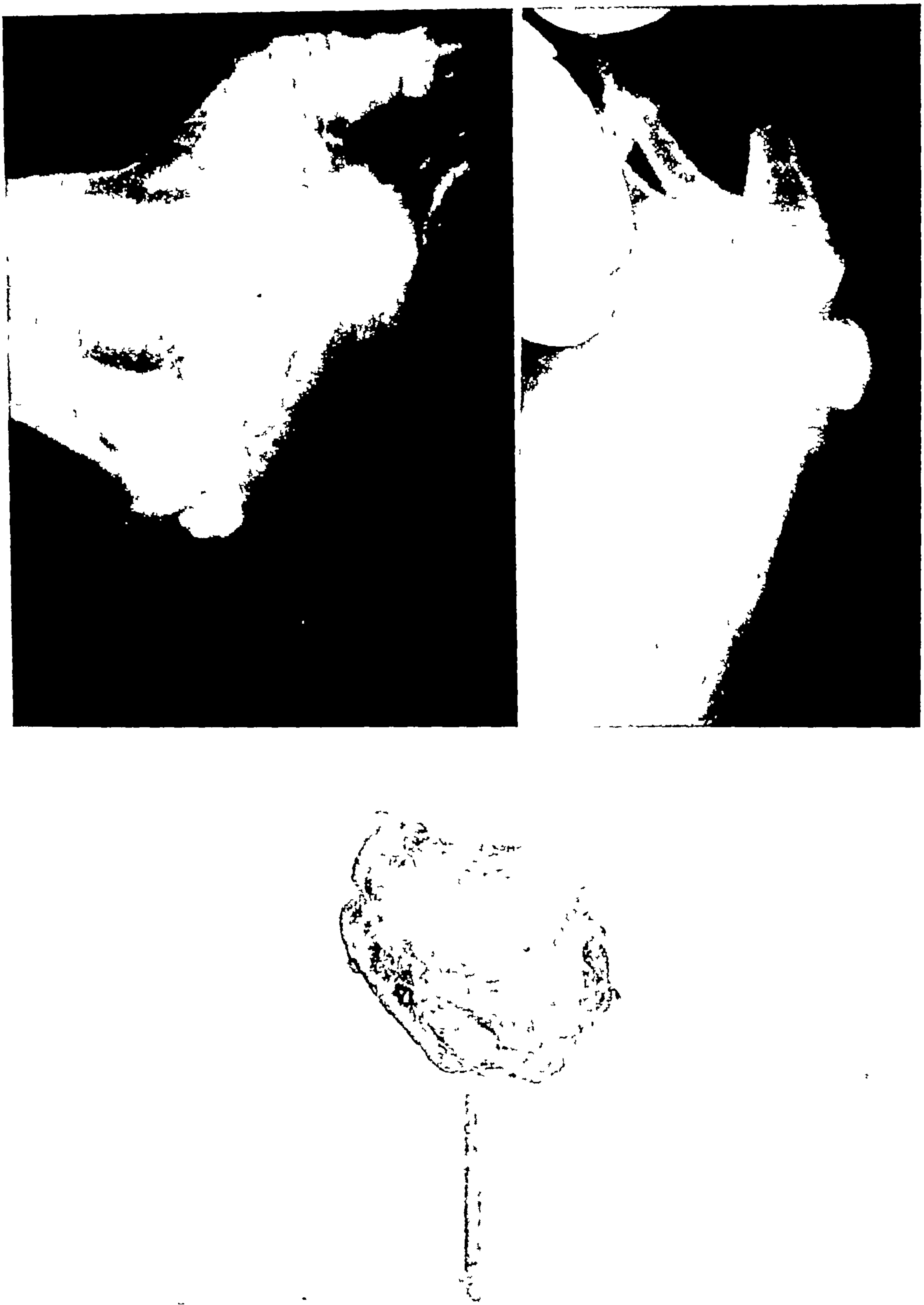
Fig 225 —Comminuted fracture of fibular sesamoid

DISTORTED AND HYPERTROPHIED SESAMOIDS

The sesamoids vary widely in size and shape Some may have projective areas which may become weight-bearing points, thereby producing a deep-seated callus, usually under the tibial sesamoid

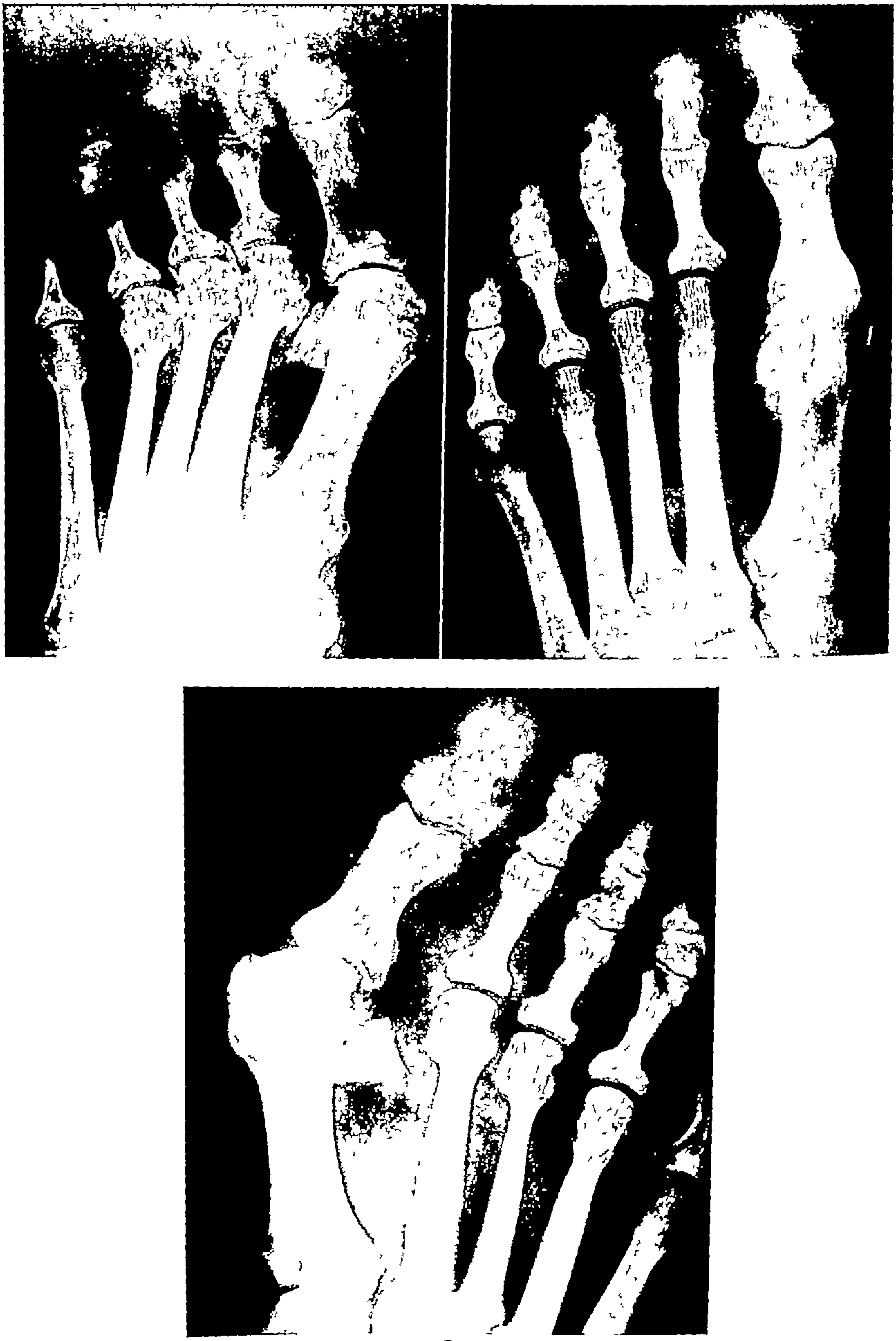
Congenital variations of the sesamoids may become a problem if the whole or part of the plantar surface of the sesamoid is not smooth or evenly shaped The types vary extensively They may be extraordinarily large or thick or have a sharp projection on the plantar surface Acquired irregularities of a sesamoid may be secondary to a congenital anomalously shaped sesamoid or to rotation

A B



C

Fig 226.—*A*, Hypertrophic ridge on plantar surface of tibial sesamoid, produced hyperkeratosis in skin beneath it *B*, Unusually thick tibial sesamoid, induced deep-seated callus beneath *C*, Extensive hypertrophy of excised sesamoid which had caused a chronic ulcer under first metatarsal head



A B

C

Fig 227 —Hypertrophic changes of fibular sesamoid, produced pain in first metatarsal interspace

of the sesamoid secondary to deformity of the great toe joint, because the sesamoids normally fit into a groove on the plantar surface of the first metatarsal head (See Hallux Valgus, page 367) Any rotation or adduction of the first metatarsal gradually tends to rotate the sesamoids

The distorted shape may also be due to hypertrophy of the sesamoids If hypertrophy takes place plantarwise, the excess bone will induce a deep-seated callus and may ultimately ulcerate the soft tissue beneath it, because of weight-bearing pressure under it (Fig 226)

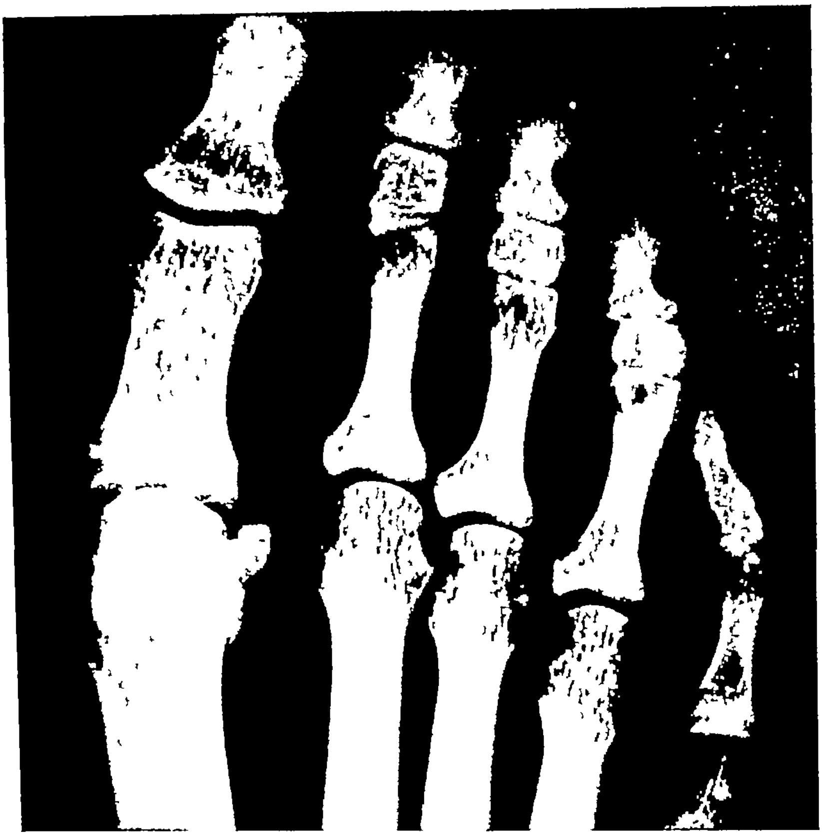


Fig 228 —Exostosis of fibular sesamoid

Symptoms.—Constant pain on weight bearing is the symptom that makes the patient seek relief A deep-seated callus is typically present under the pivotal area The callus may be mistaken for a verruca plantaris The soft tissue under the sesamoid, debilitated by chronic irritation, may ulcerate and resist all palliative treatment Misshapen or hypertrophied fibular sesamoids seldom produce keratotic changes of the skin, because, normally, they do not bear weight The pain experienced between the first and second metatarsal heads may be due to irritation (Fig 227) Occasionally an exostosis develops on the fibular sesamoid (Fig 228)

Treatment.—In mild cases proper weight distribution is alleviating In protracted cases, sesamoidectomy is indicated (See pages 194, 228 and 367)

OSTEOCHONDROSIS OF SESAMOIDS

Osteochondritis of sesamoids is not so rare as is generally assumed Caravias (1957), however, found only two publications on the subject The fibular sesa-

moid is the one likely to be affected. The condition begins as a degeneration or necrosis and is followed by regeneration and excessive recalcification Osteochondrosis may be asymptomatic and its presence disclosed only accidentally during routine examination When the disorder becomes symptomatic, pain in the first metatarsal interspace becomes chronic Symptoms, however, are self-limiting, only rarely does the sesamoid need to be excised.

I saw one case of osteochondritis of the tibial sesamoid which resulted in ultimate absorption of the entire bone

Fig 229 —Cystic degeneration of tibial sesamoid produced chronic pain

CYSTIC DEGENERATION OF SESAMOIDS

Either sesamoid may undergo cystic changes, but the tibial sesamoid is affected oftener (Fig 229) than the fibular The symptoms are chronic pain and tenderness under the first metatarsal head Excision of the sesamoid is indicated when repeated padding fails to relieve pressure of weight on the affected area

CONGENITAL ABSENCE OF TIBIAL SESAMOID

Inge (1936) reported two cases of asymptomatic congenital absence of the tibial sesamoid on one foot in each case. The anomaly is probably more prevalent than is realized It goes unnoticed probably because it is ordinarily symptomless, the condition, however, can produce a painful callus under the first metatarsal head when the great toe is hammered The absence of the sesamoid

Fig 230 — Congenital absence of both tibial sesamoids caused a hammered great toe, with resulting pain under first metatarsal head

Fig 231 — Accessory sesamoid in short flexor of second toe which rotated into first metatarsal interspace Chronic pain

weakens the flexor hallucis brevis, thereby predisposing the great toe to a clawed shape In a personal case (Fig 230), bilateral absence of the tibial sesamoid produced such deformity and intractable callus under the first metatarsal head as to require reduction of the hammered toe and lengthening of the extensor hallucis longus for relief of symptoms

INCONSTANT SESAMOIDS

Accessory or inconstant sesamoids may occur under any weight-bearing surface of the foot, especially under the heads of the lesser metatarsals or any of the phalanges, and sometimes under all the metatarsal heads (Fig 218) Patterson (1937) and Lapidus (1940) each reported such a case The accessory sesamoids

Fig 232—Large accessory sesamoid under head of first proximal phalanx caused chronic ulcer beneath

vary widely in size and shape They are ordinarily asymptomatic but may become painful when the ossicle is extraordinarily large or when the metatarsal above it rotates They are likelier to produce symptoms under the second (Fig 231) or fifth metatarsal head Under the second metatarsal head, sesamoids can be excised through an incision over the first metatarsal interspace, under the fifth metatarsal head, they are excised through an incision along the lateral plantar border of the fifth metatarsophalangeal joint

SESAMOID UNDER HEAD OF FIRST PROXIMAL PHALANX

Sesamoids as accessory bones under the head of the first proximal phalanx occur often, they are typically singular but may appear in pairs An accessory

sesamoid becomes disabling when it is unusually large or when the phalanx becomes rotated so that the sesamoid becomes a pivot This happens in some cases of hallux valgus

Characteristics.—The fibular side of the phalanx (Fig 232) is involved oftener than the tibular The disorder is first evident as a deep-seated callus which may break down and ulcerate

Treatment.—The sesamoid under the head of the first proximal phalanx can readily be excised through an incision on that side of the toe having the offending ossicle

REFERENCES

Caravias, D E Osteochondritis of Sesamoid Bones, Brit J Surg 44 623, May, 1957

Gottlieb, A Clinical Significance of Accessory Bones of Foot, West J Surg 49 452-453, Aug , 1941

Inge, G A L Congenital Absence of Medial Sesamoid Bone of Great Toe, J Bone & Joint Surg 18 188, Jan , 1936

Inge, G A L , and Ferguson, A B Surgery of Sesamoid Bones of Great Toe, Arch Surg 27 466, Sept., 1933

Kewenter, Yngve Sesamoid Bones of Human First Metatarsophalangeal Joint, A Clinical, X-ray, and Histopathological Study, Acta orthop scandinav , supp 2, 1936

Lapidus, P W Sesamoids Beneath All Metatarsal Heads of Both Feet, J Bone & Joint Surg 22 1059, Oct , 1940

Patterson, R F Multiple Sesamoids of Hands and Feet, J Bone & Joint Surg 19 531, April, 1937

Toepel, T Sesamoid Bones, Important Factors in Deformities of First Metatarsophalangeal Joint, J M A Georgia 18 499, Nov , 1929

CHAPTER THIRTEEN

Diseases of Nerves

MOST NEUROLOGIC DISORDERS OF THE FOOT STEM FROM CHANGES IN THE CEREBROSPINAL system, with secondary changes in the lower extremity Except for connective tissue changes in the digital branches of the plantar nerve, intrinsic diseases of the nerves of the foot are rare Some discussion of them, however, is pertinent here Extrinsic neurologic diseases are mentioned for inclusiveness.

All neurologic problems of the foot must be differentiated between upper and lower motor neuron disease (Fig 233) Differential diagnosis, reflexes, and gait in neurologic diseases of the lower extremities show the following characteristics

When the *lower* motor neuron is affected, the base of the step is broad and the person bears weight first on the heel, the muscles are flaccid, the tendocalcaneus reflex is diminished, electrical changes take place, there is atrophy, Babinski's sign is negative, ankle clonus is absent Anterior poliomyelitis and peripheral neuritis are the most frequent diseases producing lower motor neuron symptoms

When the *upper* motor neuron is involved, the base of the step is narrow and the person bears weight on the ball of the foot, the muscles are spastic, the tendocalcaneous reflex is increased, electrical changes do not take place, there is no atrophy, Babinski's sign is positive, and ankle clonus is present Multiple sclerosis or tumor of the brain is often the underlying disease

NEUROFIBROMA

Neurofibroma (neurinoma) is an accumulation of collagenous material of the neurilemma (sheath of Schwann) Neurilemma is present only in peripheral nerves, therefore, such changes occur only in peripheral nerves, as pointed out by both Boyd (1943) and Ewing (1942) It was Ewing who suggested that the

term *neurinoma* supplant *neurofibroma* as being more descriptive of the true pathologic condition Winkler and his associates (1948) reviewed microscopic sections of twenty specimens of presumed neurofibroma of the foot but did not find evidence of active proliferation of either nerve or neurilemma. They found instead that the enlargement was due to a deposition of hyaline and collagenous material.

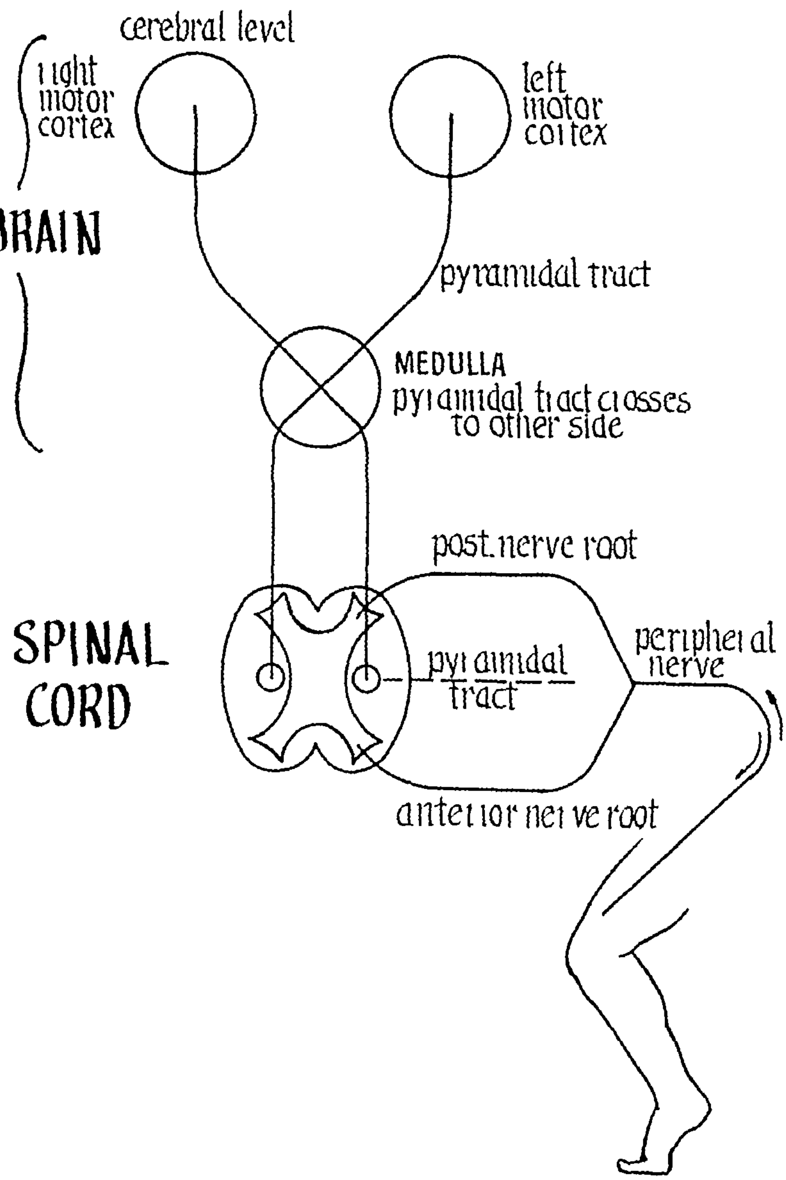


Fig 233 —Schematic drawing showing pathways of upper and lower neurons

Morton in 1876 described the entity known as *Morton's neuralgia* or *Morton's metatarsalgia* He suggested that the condition is probably caused by neuritis of the third branch of the medioplantar nerve Textbooks on anatomy describe the medioplantar nerve as having four digital branches and name them one to four Gray names the first the *cutaneous* branch to the big toe, and the rest as *first, second,* and *third digital* branches (Fig 234) The present text follows Gray's terminology

Tubby (1912) treated a series of patients who had so-called Morton's neuralgia by excising the head of the fourth metatarsal He stated "On occasion when the nerve was seen, it was resected and it often showed small nodular masses" Nissen (1948) remarked that before 1940 several English surgeons who had noticed nodular masses on the plantar digital nerve in the course of surgical procedures involving the plantar surface thought that fibrous changes were taking place Betts (1940) was the first to demonstrate histologically the extensive connective tissue enlargement of the third digital branch of the medioplantar nerve He observed this in nineteen cases in which he and his colleagues performed resections of the nerve because of so-called Morton's neuralgia McElvenny (1943) and McKeever (1952) reported independently that they found

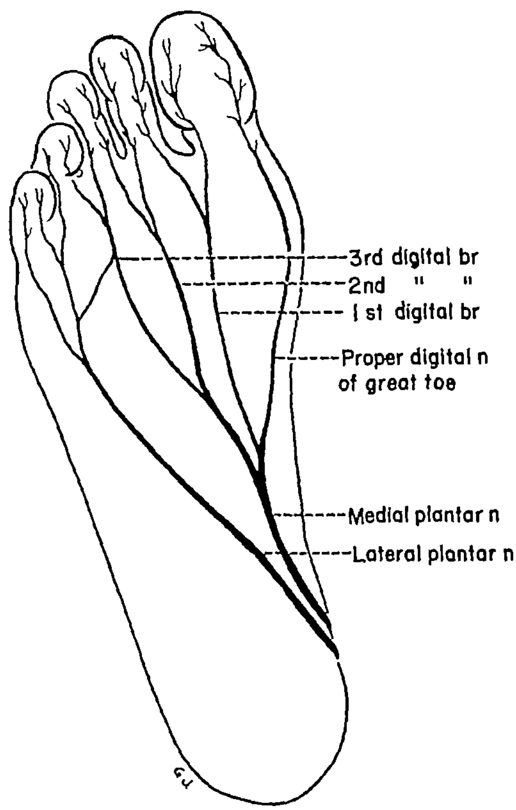


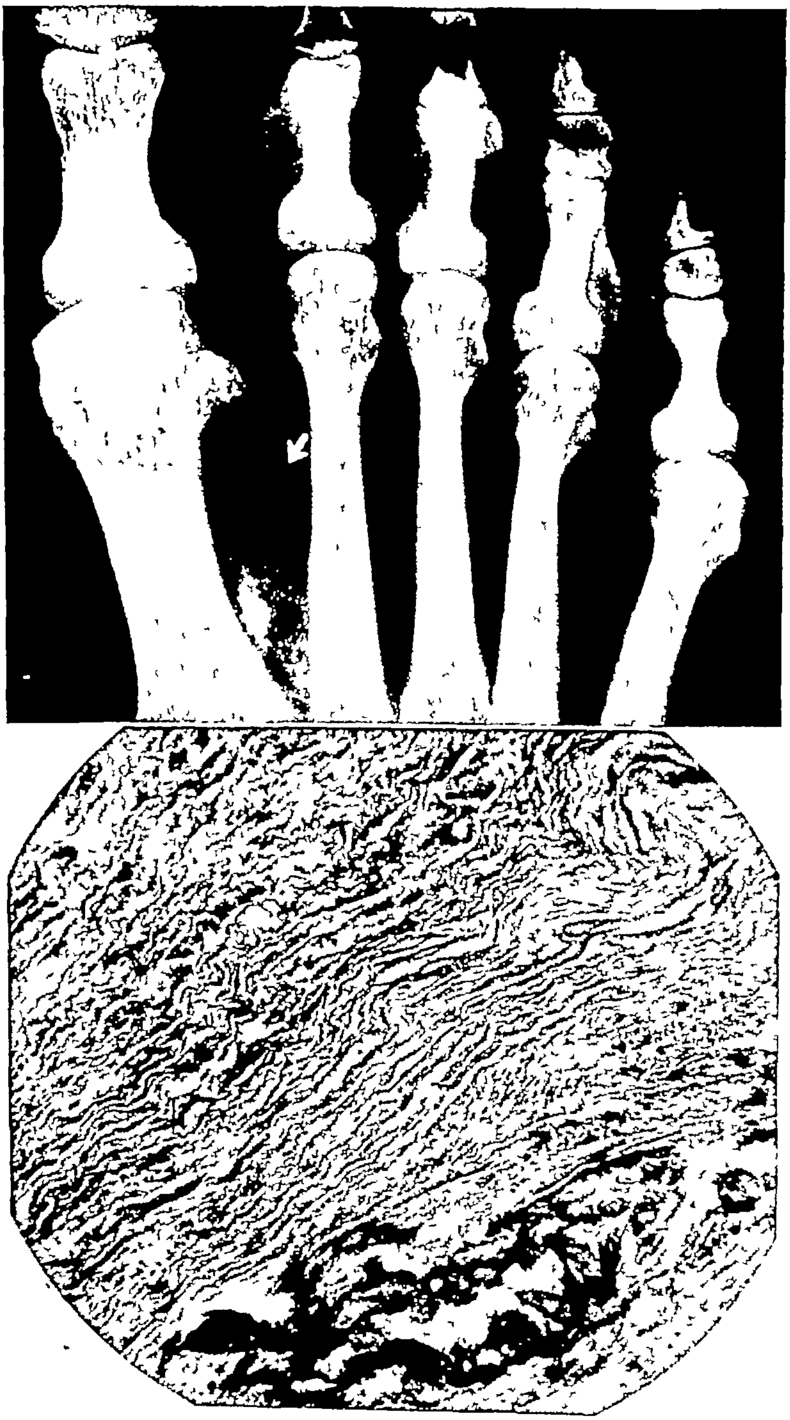
Fig 234 –Plantar nerves of right foot

the presence of a thickened digital nerve in the third metatarsal interspace about the same time that Betts did Since then, Baker and Kuhn (1944), Bickel (1947), Watson-Jones (1949), and others have substantiated Betts' observations Pincus (1950) thoroughly reviewed publications concerning neurofibroma of the foot The reported cases of neurofibroma of the foot refer to the third metatarsal interspace involving the third digital branch of the medioplantar nerve Apparently the only exception is a case reported by Hauser (1943), who described the excision of a massive neurofibroma of the first digital branch In 1952 DuVries and Cascino reported a case involving the first digital nerve The observations

in that case were confusing because the growth contained a large amount of radiopaque material (Fig 235)

Neurofibroma is somewhat more prevalent in women The ratio reported by investigators varies (Scotti, 1957), but in a personal series of 200 cases the ratio has been four to one The lesion occurs at all ages but mostly between 30 and 50 years, and especially in a narrow, flaccid type foot

A



B

Fig 235 —*A*, Calcified neurinoma of first branch of medioplantar nerve *B*, Palisade arrangement of cells typical of neurinoma, with inert deposits in lower part of field (×85)

Etiology.—The cause of neurofibroma as related to the foot is not entirely clear Nissen (1948) believes it is due to the peculiar anatomy of the third plantar digital artery It lies diagonally across the third digital nerve which may cause pressure on the artery and the nerve when the patient bears weight Winkler and his associates (1948) postulate that the predisposition of the third digital nerve to the disease is related to its receiving a filament from the latero-plantar nerve (Fig 234), although the third digital nerve is essentially a branch of the medioplantar nerve It is possible that neurinomas of the digital plantar nerve branches result from chronic irritation Irritating factors include the wearing of faulty footwear, walking on hard floors, standing for long periods, and anatomic variations of bones and nerves on the plantar surface, which combine variously to produce neurinomas Prolonged irritation of living tissue, whether caused by chemical agents or mechanical friction, induces proliferation of connective tissue or collagenous material in the involved area The three digital branches of the medioplantar nerve lie immediately under and between the heads and shafts of the metatarsals and are subject to constant friction In response to friction, collagenous connective tissue accumulates in the neurilemma Increased thickness subjects it to greater friction The third digital nerve is by far most frequently affected The second digital nerve sometimes is affected and only rarely, the first digital nerve The overwhelming predilection to thickening of the third digital branch over the other two branches is probably due to the peculiar anatomic structure of the fourth metatarsal Of the five metatarsals, the fourth is least securely anchored at its base (Fig 32) This permits a floating action of the distal end of the fourth metatarsal when the foot is in motion The anatomic instability of this metatarsal head may well cause a constant grating against the terminal portion of the third digital nerve

Dissection and sectioning of many digital medioplantar nerves, taken at random from cadavers and freshly amputated specimens, show comparable microscopic data in neurofibromas

Symptoms—When the second or third branch is affected, the early symptoms are similar to simple metatarsalgia (Morton's neuralgia) varying degrees of pain about the fourth metatarsophalangeal joint and in the third metatarsal interspace, which is aggravated by weight bearing The symptoms gradually worsen and some cases become intractable In simple metatarsalgia, pain is experienced only on weight bearing, whereas in neurinoma, pain is constant, even when the foot is at rest

Diagnosis—When the interspace is palpated, the patient experiences a sharp stabbing pain Palliative measures, such as padding or the use of a Thomas bar, relieve simple metatarsalgia but do not give relief from symptoms of neurinoma, thus, to some degree, palliative relief offers differential diagnosis

The growth is ordinarily too small to be palpated (Fig 236) The diagnosis must be made on the basis of clinical observations, however; neither roentgenograms nor laboratory tests offer clues Intractable pain in the interspace between the third and fourth metatarsal head, which cannot be relieved by mechanical measures, justifies a diagnosis of operable neurinoma.

Neurinoma of the first digital nerve, because it is massive, is readily palpated immediately under the first metatarsal interspace in which it occurs Pain is at least as severe as in the lesser branches

Treatment.—Excision of the entire nerve gives complete relief. European surgeons excise the nerve of the second or third digital branch through a plantar approach, as described by Betts (1940) and by Nissen (1948) In this country the approach generally is through the dorsum, as described by McElvenny (1943) and by McKeever (1952) The American approach has advantages, the most important being prevention of scar formation on the plantar surface Furthermore, this approach does not pass through important fascial planes as does the plantar incision The nerve is easily pressed into the metatarsal interspace and does not present obstacles to grasping and excision

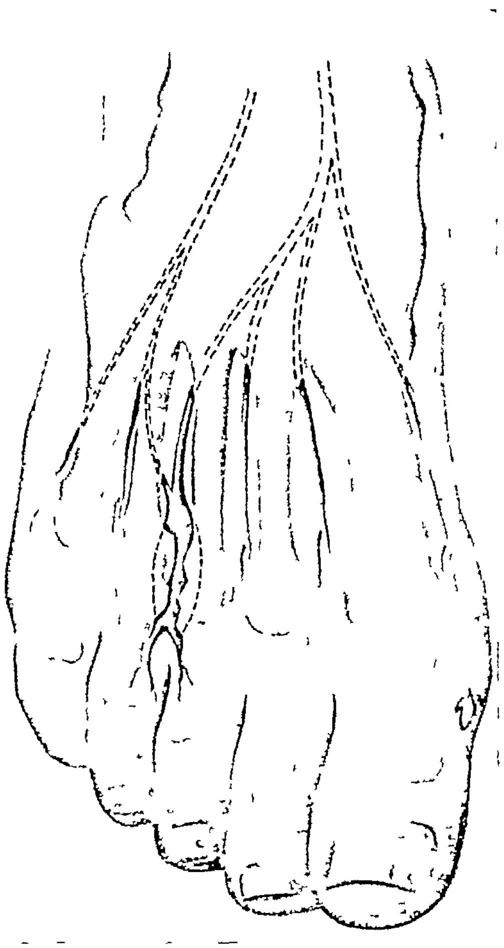


Fig 236 —Neurinoma of third branch of medioplantar nerve, shown schematically

Operative Technique for Neurinoma Commonly Involving Second or Third Digital Nerve —In twenty-five referred surgical cases of presumed neurinoma of the digital branch of the lateroplantar nerve in the fourth metatarsal interspace, the pathologist reported that the characteristic palisade arrangement of neurinoma was absent In two cases, the pathologist observed glomus cells, indicating a glomus tumor Operation did relieve symptoms in most cases

1. Under hemostasis, make an incision over the dorsum of the metatarsal interspace, extending it from the center point in the web of the toes proximally to about the middle of the metatarsal interspace

2 Carry the incision down into the lower depth of the interspace

3 Spread apart the heads of the adjacent metatarsals by means of a Weitlander or mastoid retractor.

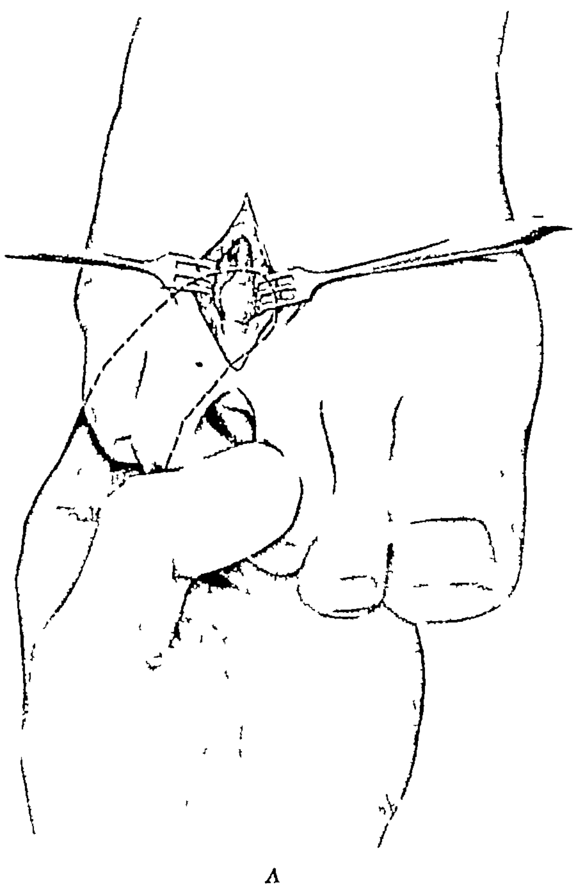
4 On application of pressure with a finger on the plantar surface of the metatarsal interspace, an encapsulated lobular mass bulges into the interspace (Fig 237, *A*). Grasp the mass, which includes the nerve, with Allis forceps, and begin its excision at its distal end, continue proximally until its pedicle escapes under the plantar surface of the base of the metatarsals, there, excise it (Fig 237, *B*)

5 Instill hydrocortisone, 5 mg, in the wound.

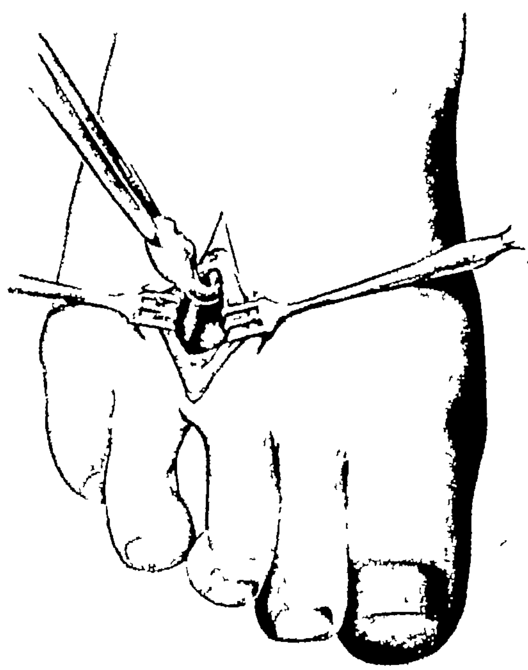
6 Suture the skin and fascia in layers

7. Apply a compression bandage, and release it in twenty-four hours, at which time ambulation may begin.

The procedure is the same when the lesion is in the second metatarsal interspace



A



B

Fig 237 –*A*, By applying pressure with index finger against distal end of third metatarsal interspace, neurinoma comes into view *B*, Neurinoma dissected proximally as far as possible and brought out of wound, amputated at its most proximal end

Operative Technique for Neurofibroma Involving First Digital Nerve — The approach is best through a horizontal medioplantar incision, extending proximally from a point just behind the first metatarsal head to the medial cuneiform

1 Retract the plantar margin of the incision so as to expose the medial margin of the plantar fascia

2 Incise the fascia horizontally and deflect plantarwise

3 At the distal angle of the fascia, a mass begins to bulge at this stage

4 Grasp this mass and dissect proximally until it becomes a thin pedicle (Fig. 238), then sever it at the neck

5 Instill about 10 mg hydrocortisone into the wound

6 Close the fascia and skin in layers and apply a compression bandage. Ambulation may begin in twenty-four hours after compression has been released. Postoperative pain is moderate, complete recovery is rapid.

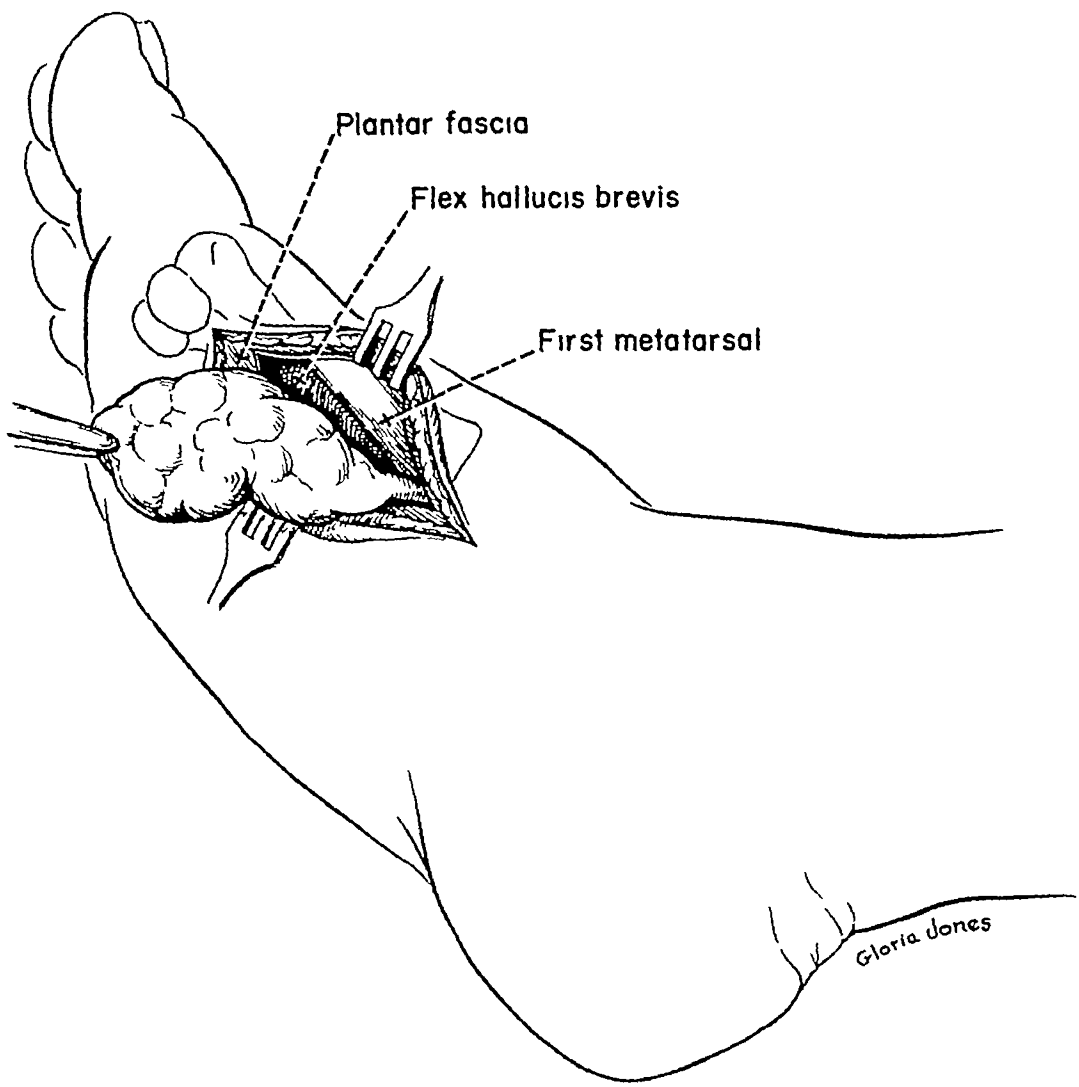


Fig 238 —Delivery of neurinoma of first branch of medioplantar nerve

CAUSALGIA

Causalgia is a neuralgia characterized by constant intense local pain and burning, which may be accompanied by local skin changes, such as redness and vesicle formation It is a clinical syndrome associated with lesions of the nerves, resulting from accidental or surgical trauma to a peripheral nerve containing sensory fibers

Etiology.—Causalgia results when a nerve trunk is incorporated in a surgical or fracture scar, or when a sensory nerve receives a direct injury, as by a gunshot wound. The pain is over or distal to the area of the nerve lesion and corresponds to cutaneous distribution of the nerve Pressure or palpation of the area where the nerve is included in a scar accentuates the pain The region may show trophic and vasomotor changes In amputees, the pain is experienced in the region of the stump or as *phantom pain* in the structures already amputated

With reference to the foot, causalgia is in most cases a postoperative complication De Tâkats (1945) calls attention to other painful conditions in the extremities from injuries to the peripheral nerve Cullen (1948) reported twenty-four cases of causalgia, five involving the foot Severe osteoporosis, resembling Sudeck's atrophy, developed in all the bones of the foot in one patient twelve months after injury Causalgia in all five cases resulted from injuries to the internal popliteal nerve The distribution of pain in all instances was in the sole of the foot. In the cases personally observed, causalgic pain was along the course of the laterodorsal cutaneous nerve from the lateral malleolus to the fifth toe The laterodorsal cutaneous nerve in that area is superficial, so that its inclusion in a scar along its course is understandable Stroking the area gave rise to an unbearably sharp burning sensation, mainly distal to the nerve lesion

Treatment.—The treatment discussed here relates only to the foot Injection of procaine hydrochloride or alcohol has been recommended, but the results are doubtful Roentgen therapy has also been suggested, but its efficacy has not been proved In cases of causalgia of the foot due to severe injury to the posterior tibial or common peroneal nerve, parasympathetic block is a valuable therapeutic measure as well as a diagnostic and prognostic test, for it foretells whether sympathectomy will give permanent relief

When causalgia is due to direct injuries of the foot, neurolysis or neurectomy offers best results The sensory nerve trunk involved should be freed of all entanglements of scar tissue If that is not possible, the trunk should be exposed proximally to the area where it is entwined in scar tissue and about 1 cm of the nerve excised

Technique to Free Nerve From Scar—Good surgical exposure is essential to any attempt at freeing a nerve from a scar and requires, therefore, general anesthesia and complete hemostasis

1 Excise all scar tissue or callus surrounding the nerve

2 Surrounding the freed nerve by a bed of fat obtained from an adjacent area

3 Carefully close the fascia and skin in layers *Caution* Do not entwine the nerve in a suture

4. A compression bandage applied for twenty-four hours prevents excessive bleeding into the wound after hemostasis has been discontinued

5. In cases in which a nerve cannot be located and in recurrent cases, excise about 1 cm of the nerve trunk proximally to the original injury Cover the proximal end of the severed nerve and embed it in fat.

Denervation of Ankle to Relieve Intractable Pain —Although neurectomy has been used for many years in cases of intractable pain, notably of the hip joint as described, for example, by Mulder (1948), denervation of the ankle is a relatively new procedure. Casagrande and co-workers (1951) were probably the first to call attention to the simplicity and value of denervation of the ankle in cases of traumatic arthritis not amenable to other forms of therapy for relief of pain Their article gives a more detailed description of the nerve distribution around the ankle than is available in standard textbooks on anatomy They advised sectioning the articular branches of the posterior tibial and deep peroneal nerves, including the terminal branch of the deep peroneal nerves Only negligible motor loss in the extensor brevis digitorum results from sectioning the deep peroneal nerve

In *Casagrande's technique,* under hemostasis and general anesthesia, an incision is made about 1 5 cm behind the medial malleolus and is carried downward, then curved anteriorly around the tip of the malleolus so as to form a reverse J-shaped incision The incision is carried farther down to the laciniate ligaments The tibial nerve lies about 2 cm beneath the skin, lateral to the flexor digitorum longus tendon Directly anterior and superficial to the nerve are the posterior tibial vessels, which are contained with the nerve in the neurovascular sheath The articular branches can be identified as they pierce the deltoid ligament just above the division of the tibial nerve into the mediolateral and lateroplantar nerves The articular branches are ligated and sectioned The approach to the deep peroneal nerve is through an S-shaped incision on the anterior skin surface of the ankle, just lateral to the extensor hallucis longus tendon The annular ligament is incised longitudinally and retracted The deep peroneal nerve may be identified between the tendons of the extensor hallucis longus and extensor digitorum longus Articular filaments can be identified just above the bifurcation of the nerve and into the medial and lateral terminal branches They are ligated and sectioned, the lateral terminal branch is also ligated and sectioned to ensure complete denervation The skin and fascial structures are closed in layers. Complete closure of the annular ligament is essential, because it maintains the extensor tendons as they leave the ankle and enter the foot

NEUROPATHIES

The neuropathies comprise a group of syndromes, sometimes referred to as arthropathies, as described by Charcot in 1868, whose chief characteristic is massive bone destruction as a result of neurotrophic degeneration of the spinal cord It was formerly believed that most cases were due to syphilis and only at times to such a trophic disease as syringomyelia, pernicious anemia, or diabetes Today, diabetes is the leading cause of this destructive process Although the neuropathic syndrome is a part of a general disease process, it bears description here because the foot is predominantly the area undergoing destruction

Diabetic Neuropathy

The neuropathic phase of diabetes is a specific degeneration closely resembling the neuropathic changes of latent syphilis, and, like diabetic gangrene, it has a predilection for the foot Cram (1955) refers to the syndrome as *pseudotabes,* because the changes produced in the reflexes are similar to those in syphilis The severity of diabetes does not appear to be directly associated with trophic changes, however, most of the cases reported were of long-standing diabetes Changes are essentially due to destruction of the posterior horns of the spinal cord The most drastic results of the trophic changes are complete collapse and distortion of tarsometatarsal articulation

Fig 239 —Diabetic neuropathy of tarsal joints

Although first described by Calvé as far back as 1864, few early publications appeared on the subject of diabetic neuropathy Later reports indicate that the syndrome is not uncommon, for example, Rundles (1945, 125 new cases), Bailey and Root (1947), Parsons and Norton (1951), Sheppe (1953), Antes (1954), Martin (1954), Lippman and Grow (1955), Bolen (1956), Goodman (1953)

Symptoms.—The onset is gradual, although development of osteolytic changes in the bone may be rapid, the foot becoming deformed in a comparatively short time The remnants of the metatarsals are displaced laterally (Figs 239 to 241) The foot is greatly misshapen and distorted, and there are both osteolytic and osteophytic changes Often deposition of new bone is an accompaniment, as it is in Charcot's joint Relative or complete absence of pain during the process of bone degeneration is typical, but the patient notices that his foot is

becoming unstable and its shape is changing rapidly. Sensory changes are hyperesthesia, paresthesia, and variations in degrees of pain and temperature

Pathogenesis.—Most changes in the foot are of Charcot's type of destruction at the tarsometatarsal articulation The articular surfaces are destroyed at the same time that hypertrophic changes take place, often forming loose bodies Relaxation or destruction of the ligaments results in distortion of the joints from the weight of the body

Fig 240

Fig 241

Fig 240 —Diabetic neuropathy, with complete collapse of the tarsometatarsal articulations

Fig 241 —Diabetic neuropathy of tarsometatarsal articulations Previous episode of diabetic gangrene of fifth toe

Treatment.—Only recent changes are to some extent reversible In advanced cases destruction is so extensive that surgical intervention is of little benefit Stability can be improved by use of metal inlays, pads, high shoes, or leg braces In many instances, amputation is advisable, because of the likelihood of diabetic ulcers, leading to gangrene

Syphilitic Neuropathy

Until the advent of antibiotics, latent syphilis was the usual destroyer of the joints of the body (Figs 242 and 243) Charcot's joint due to syphilis was frequently encountered, but its predilection was for the knee rather than the foot,

Fig 242 —Necrosis of fourth digit in latent syphilis

A

B

Fig 243 —A, Charcot's joint of second metatarsophalangeal joint in latent syphilis B, Knee joint in same case Bizarre destruction and new bone formation

although the ankle was often affected and other joints were not unaffected. Antibiotics and antianemic therapy have greatly diminished the incidence of syphilitic and anemic arthropathies

Hereditary Sensory Neuropathy

A rare anomaly of the spinal cord, characterized by severe trophic disorders, is hereditary sensory neuropathy. The defect is in the dorsal root ganglion and causes insensitivity to pain. Trophic changes of the extremities are comparable to spina bifida occulta. Heller and his associates (1955) reported three cases and studied the family previously studied by Heller and Robb (1955). The disorder was traced through six generations Their own patients had trophic ulcers of the foot, loss of a foot, and neuropathic arthropathy of a foot

Neuropathy Due to Pernicious Anemia

Impairment of the peripheral nerves or generalized neurogenic disease is a complication of pernicious anemia When the peripheral nerves are impaired or degenerate, changes in the foot result, often leading to extensive osteolysis of the bones. In a personal case, destruction worsened, notwithstanding intensive antianemic therapy (Fig. 244)

Spondylolisthesis

Spondylolisthesis (Jenkins, 1936, Meyerding, 1931, 1956) is a forward displacement of the fifth lumbar vertebrae and spinal column from the sacrum Occasionally the displacement is of the fourth lumbar vertebrae from the fifth lumbar vertebrae (Chandler, 1931) The anomaly is usually due to a developmental defect in the neural arch Absence of patellar and tendo achillis reflexes and diminished sensory response should suggest the need for roentgenograms of the spine In a personal case (Fig 245), a mediolateral view of the lower part of the spine disclosed severe spondylolisthesis and cord distortion Laminectomy to free the cord roots and spinal fusion resolved the neurologic symptoms, which began with the patient's experiencing tenderness and observing induration of the skin on the plantar surface of the left heel A wheal formed which had been mistakenly diagnosed as a dermatologic disorder

Spina Bifida in Relation to Lower Extremities

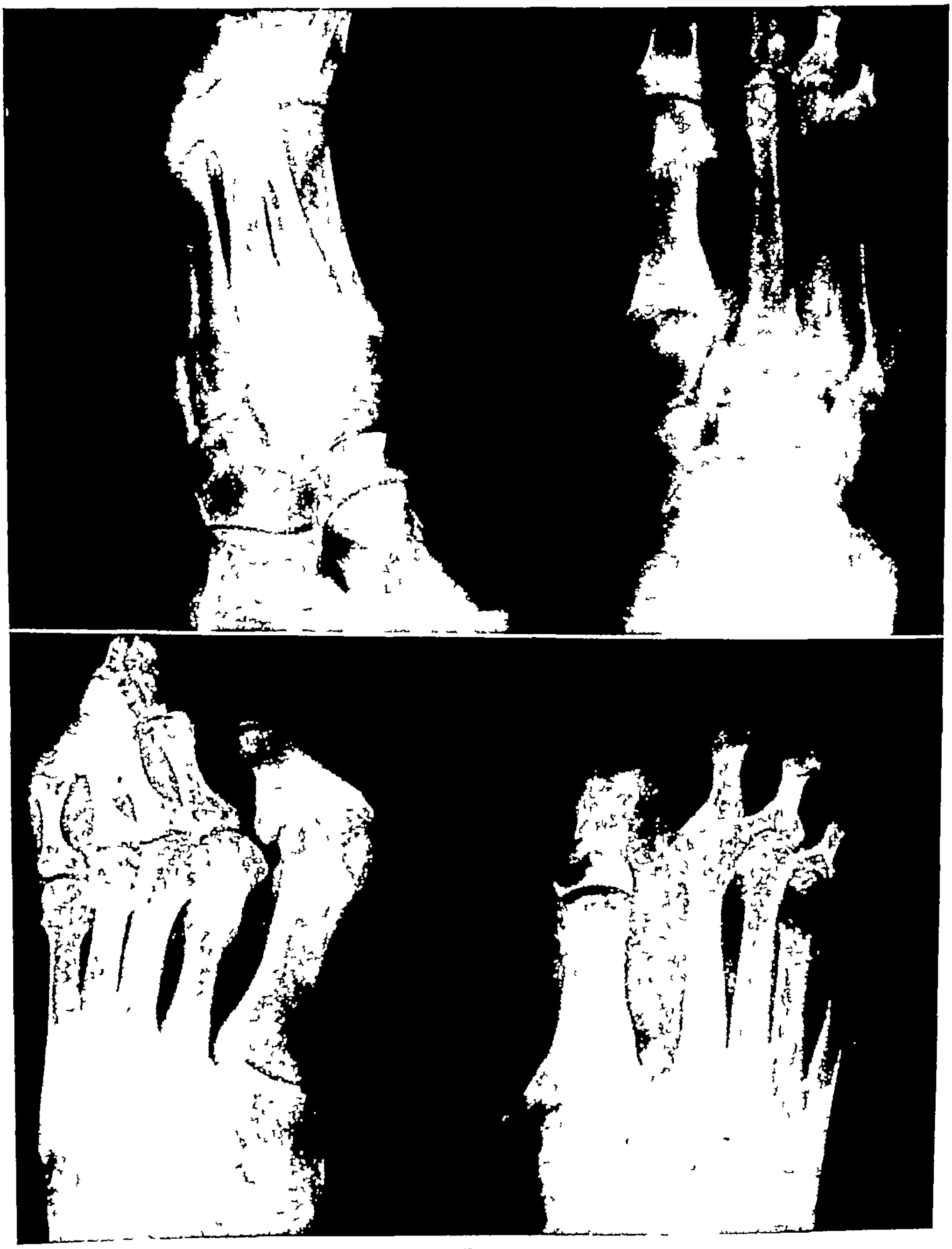
Spina bifida is a congenital defect of the vertebral column, varying from a simple defect in the spinous process to extensive clefts and malformation of the entire arch of one vertebra or several vertebrae (rachischisis), thereby leaving the spinal cord exposed, and frequently resulting in trophic changes and deformities of the lower extremities (Fig 246)

Pathogenesis.—The disorder is primarily a defective formation of the neural arches, usually of the fifth lumbar and sacral vertebrae, less frequently of those of the upper lumbar, and only rarely of the dorsal or cervical section The defect

is covered by a tough connective tissue membrane, which is usually adherent to the spinal cord

Complications in Lower Extremity —There have been many publications evidencing the relation of spina bifida occulta to the feet (Mixter, Woltman, Brunner, von Recklinghausen, Bibergeil, Gillies and Hartung) Syndactylia and frequent motor disturbances have been observed, especially clawfoot and

A



B

Fig 244 —A, Arthropathy of first metatarsal-first cuneiform joint in latent pernicious anemia B, Three months later Further destruction of head of the right first metatarsal

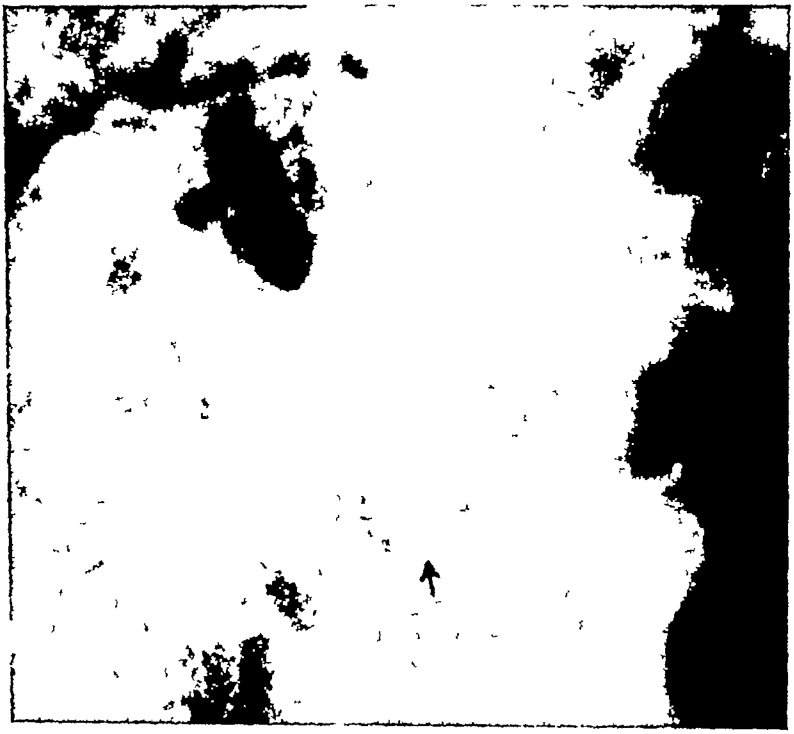


Fig 245 —Spondylolisthesis of fifth lumbar vertebra First symptoms pain and induration on plantar surface of left heel

Fig 246 —Defects of lumbosacral vertebra in case of spina bifida

A

B.

Fig 247 —Same case as shown in Fig 246 *A*, Deformity of feet *B*, Trophic destruction of first metatarsophalangeal joints

talipes in any attitude, drop foot or drop toe, and always an abnormal gait Among the trophic disturbances, perforating ulcer of the foot is the most usual (Fig 247). It results from muscular imbalance and contracture, coupled with the characteristic low vitality of the soft structures. All the complications of trophic degeneration are possible osteomyelitis, severe phlegmonous cellulitis of deep structures, probably as a result of infection which may be incurred during minor surgical procedures, septicemia, and necrosis Lowered sensory perception may lead to neglect of pressured points for which people in pain seek relief Sensory disturbances have been observed along the distribution of the sciatic nerve, back of the thigh, and at the posterior of the calf, covering the field of distribution of the fifth lumbar and first, second, and third sacral roots In other cases in which the lower sacral roots are involved, there is a typical saddle block anesthesia (Steindler) Hypoesthesia may be present in all forms and, sometimes, paraplegia

Treatment.—Attempts to distribute weight bearing must always be made, but it should be kept in mind that a new weight-bearing area may become a focal point for trophic ulcer. Weight bearing must be as restricted as can be managed

Cleanliness of the foot is essential, as are appropriate shoes to accommodate the particular deformity among various types of deformities in such cases

REFERENCES

Antes, E H Charcot Joint in Diabetes Mellitus, J A M A 156 602, Oct 9, 1954

Bailey, C C, and Root, H F Neuropathic Foot Lesions in Diabetes Mellitus, New England J Med 236 397, March 13, 1947

Baker, L D, and Kuhn H H Morton's Metatarsalgia, Localized Degenerative Fibrosis With Neuromatous Proliferation of the Fourth Plantar Nerve, South M J 37 123, March, 1944

Ballantine, H T Practice of Surgery, Hagerstown, Md, 1955, W F Prior Co, Inc, vol 12, chap 3

Betts, L O Morton's Metatarsalgia, M J Australia 1 514 April 13 1940

Bickel, W H, and Dockerty, M B Plantar Neuromas, Morton's Toe, Surg Gynec & Obst 84·111, Jan, 1947

Bibergeil, E Die klinische Bedeutung der Spina bifida occulta, Klin Wchnschr 1 1481-1484, 1913

Bolen, J G Diabetic Charcot Joints, Radiology 67 95, July, 1956

Boyd, W Textbook of Pathology, ed 4, Philadelphia, 1943, Lea & Febiger, p 906

Brunner, J C Fall von Spina bifida occulta, Virchows Arch path Anat 107 194, 1887

Calvé, M de Recherches sur les Accidents Diabétique, Paris, 1864 P Asselin

Casagrande, P A, Austin, B P, and Indeck, W Denervation of Ankle Joint, J Bone & Joint Surg 33A 723, July, 1951

Chandler, F A Lesions of "Isthmus" (Pars Interarticularis) of Laminae of Lower Lumbar Vertebrae and Their Relation to Spondylolisthesis, Surg Gynec & Obst 53 273-306, Sept, 1931

Charcot, J M Sur Quelques Arthropathies, du Cerveau on de la Molle Epinière, Arch Physiol 1 161-178, 579-599, Jan, Feb, 1868

Cram, R H Orthopaedic Aspects of Diabetic Neuropath, J Bone & Joint Surg 37A 967, Oct, 1955

Cullen, C H Causalgia (Diagnosis and Treatment), J Bone & Joint Surg 30B 467, Aug, 1948

de Takats G Causalgic States in Peace and War, J A M A 128 699, July 7, 1945

DuVries, H L, and Cascino, J P Massive Neurofibroma (Neurominoma) of the Foot With Calcification J Internat Coll Surg 17 688, May, 1952

Ewing, J Neoplastic Diseases, ed 4, Philadelphia, 1942, W B Saunders Co

Gillies, C L, and Hartung, W Fracture of Tibia in Spina Bifida, 2 Cases, Radiology 31 621-623, 1938

Goodman, J I, Siegfried, B, Frankel, L, Marcus, L J, and Wasserman, S Diabetic Neuropathies, Springfield, Ill, 1953, Charles C Thomas, Publisher
Gray, Henry Anatomy, ed. 26, Philadelphia, 1954, Lea & Febiger
Hauser, E D W Neurofibroma of the Foot, J A M A **121** 1217, April 10, 1943
Heller, I H, and Robb, P Hereditary Sensory Neuropathy, Neurology **5** 15, Jan, 1955
Heller, L, Heller, I H, and Petrie, J G Orthopedic Problems of Hereditary Sensory Neuropathy, J Bone & Joint Surg **37B** 632, Nov, 1955
Jenkins, J A Spondylolisthesis, Brit J Surg **24** 80-85, July, 1936
Lippman, E M, and Grow, J L Neurogenic Arthropathy Associated With Diabetes Mellitus, J Bone & Joint Surg **37A** 971, Oct, 1955
Martin, M M Neuropathic Lesions of the Feet in Diabetes Mellitus, Proc Roy Soc Med **47** 139, Feb, 1954
Meyerding, H W Spondylolisthesis, J Bone & Joint Surg **13** 39-48, Jan, 1931
Meyerding, H W Spondylolisthesis, J Internat Coll Surg **26** 566, Nov, 1956
McElvenny, R T Etiology and Surgical Treatment of Intractable Pain About the Fourth Metatarso-Phalangeal Joint (Morton's Toe), J Bone & Joint Surg **25** 675, July, 1943
McKeever, D C Surgical Approach for Neuroma of Plantar Digital Nerve (Morton's Metatarsalgia), J Bone & Joint Surg **34A** 490, April, 1952
Morton, T G Peculiar and Painful Affection of Fourth Metatarso-Phalangeal Articulation, Am J M Sc **71** 37, Jan, 1876
Mulder, J D Denervation of Hip Joint in Osteoarthritis, J Bone & Joint Surg **30B** 446, Aug, 1948
Nissen, K I Plantar Digital Neuritis, J Bone & Joint Surg **30B** 84, Feb, 1948
Parsons, H, and Norton, W S Management of Diabetic Neuropathic Joints, New England J Med **244** 935, June 21, 1951
Pincus, A Intractable Morton's Toe (Neuroma), J Nat A Chiropod **40** 19, Dec, 1950
Rundles, R W Diabetic Neuropathy, General Review With Report of 125 Cases, Medicine **24** 111, May, 1945
Scotti, T M The Lesion of Morton's Metatarsalgia (Morton's Toe), A M A Arch Path **63** 91-102, Jan, 1957
Sheppe, W M Neuropathic (Charcot) Joints Occurring in Diabetes Mellitus, Ann Int Med **39** 625, Sept, 1953
Steindler, Arthur Diseases and Deformities of Spine and Thorax, St Louis, 1929, The C V Mosby Co
Tubby, A H Deformities Including Diseases of Bone and Joint, ed 2, vol 1, London, 1912, The Macmillan Co, p 721
von Recklinghausen, Friedrich Virchows Arch path Anat **205** 262, 1886
Watson-Jones, Reginald Leri's Pleonosteosis, J Bone & Joint Surg **31B** 560-571, Nov, 1949
Winkler, H, Feltner, J B, and Kimmelstiel, P Morton's Metatarsalgia, J Bone & Joint Surg **30A** 496, April, 1948
Woltman, H W Mayo Clinic Papers, vol 12, 1920

CHAPTER FOURTEEN

Aseptic Necroses; Osteochondritis; Atrophy of Bone; Arthritides

MANY DISABILITIES OF THE FOOT INVOLVE BONE TISSUE, BECAUSE THE FOOT IS essentially an osseous structure The rigid, solid appearance of bones may disguise active and continuous alterations in their structure Nutritional, endocrinologic, and systemic diseases constantly influence the bones Although bone is a skeletal and supporting organ, the marrow within it is a blood-forming organ

Dense bone forms the shaft of long bones, the outer and inner tables of flat bones, and the outer shell of short bones *Cancellous* bone forms the epiphysis and interior of the shaft of long bones as well as the interior of short and flat bones *Periosteum* is a vascular membrane covering bones, it ends where the epiphysis joins the diaphysis The medullar portion, the marrow, is highly vascular, it is a lymphatic mass of cellular tissue inside cancellous bone and in the shaft of the long bones The nutrient artery, vein, and lymphatic vessels pierce the shaft and end in the medullary substance Periosteal vessels supply the periosteum and send inward branches to the haversian canals The minute channels in bone, which contain an artery and vein, lymph vessels, nerves, and marrow, are called the *haversian system.*

ASEPTIC NECROSES

ASEPTIC NECROSIS IN GAUCHER'S DISEASE

Gaucher's disease is a disturbance in fat metabolism Changes in the spleen, blood, and skeletal system are of unknown causation The marrow contains typical Gaucher's cells in this form of aseptic necrosis In 1948 Arkin and Schein recorded observations relating to aseptic necrosis as an important contributory factor in Gaucher's disease and repeated their earlier hypothesis that Gaucher's

Fig 248 —Aseptic necrosis of third middle phalanx

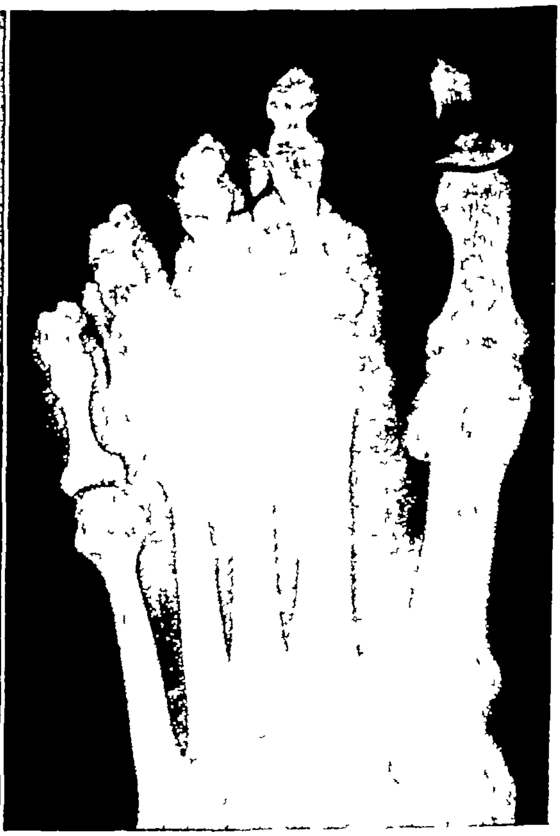


Fig 249 Fig 250

Fig 249 —Aseptic necrosis of fourth and fifth metatarsal heads (Courtesy Dr James Brightwell)

Fig 250 —Aseptic necrosis of second and third metatarsal heads of undetermined origin

disease in adults might begin as a type of osteochondrosis of childhood. These investigators believed that the infiltration of Gaucher's cells interferes with circulation and thereby causes aseptic necrosis. The disease may involve the foot Pain and tenderness of the bones and joints are experienced There may be limitation of joint movement Acute pain may suggest a diagnosis of osteomyelitis Roentgenograms of bones have areas of rarefaction and calcification which give a mottled appearance, long bones appear thickened and clublike.

ASEPTIC NECROSIS OF PHALANGES

Occasionally a case of necrosis of one or more phalanges is encountered in which a definite cause cannot be found The disease is not associated with trauma, infection, or metabolic disease It is probably due to a local interference of the blood supply to the phalanx A hereditary tendency has been observed Shaw (1954) traced the disease through six generations in one family The onset is likely to be insidious, beginning with a fusiform swelling over the phalanx involved and accompanied by moderate pain Necrosis may continue until one or more of the phalanges are completely destroyed (Fig 248)

A comparable necrosis is occasionally encountered on the metatarsal heads (Figs 249 and 250).

No rational therapy or means of arrest of the disease has been proposed Fortunately, the disease is self-limiting, the symptoms are seldom disabling

OSTEOCHONDRITIS

Osteochondritis dissecans comprises a group of diseases often classified under *osteochondroses* but variously termed Legg-Calvé-Perthes disease *(osteochondritis deformans juvenilis)* of the head of the femur is classical The disease at the outset is in reality essentially an aseptic necrosis of a portion of the articular cartilage and usually of a section of subchondral bone, most frequently in weight-bearing bones, especially those subject to prolonged trauma Although the disease begins as aseptic necrosis, it gradually changes to osteosclerosis and osteophytosis

The sites of occurrence of osteochondritis of the lower extremities have been gathered from first reports by Schaefer and associates (1939), epiphysis of calcaneus (Haglund, 1907), tibial tubercle (Osgood, 1903, Schlatter, 1904), head of femur (Legg, Calvé, Perthes, 1910), patella and tarsal navicular (Kohler, 1908), talus (Mouchet, 1929), head of second metatarsal (Freiberg, 1914), head of fifth metatarsal and tibial sesamoid of first metatarsal

KÖHLER'S NO. 1 DISEASE AND FREIBERG'S INFRACTION

Two forms of the disease in the foot have long been recognized Kohler's No 1, which affects the tarsal navicular, and Freiberg's so-called *infraction*, which affects the head of the lesser metatarsals, usually the second The disease may affect any bone of the foot Reports indicate that next to the second metatarsal head, the talus is as often affected as any bone of the foot.

Freiberg's Infraction

The term *infraction* means an incomplete fracture, it is not strictly applicable The condition is sometimes referred to as *Kohler's disease No 2* In Europe the condition is often referred to as *Panner's disease* It affects mostly the second metatarsal head, rarely the lesser metatarsal heads I have seen four cases involving the third metatarsal head (Fig 251) Breitenfelder (1937) reported a case involving the first metatarsal head The disease begins during adolescence as an aseptic necrosis with loss of bone substance of the epiphyseal head of the metatarsal (Fig. 252) During the active part of the disease there are pain and swelling about the second metatarsal head. The disease is subacute or chronic and is self-limiting The degenerative process may continue for a year or longer, followed by a regenerative process often culminating in massive osseous hypertrophy which becomes static (Fig 253)

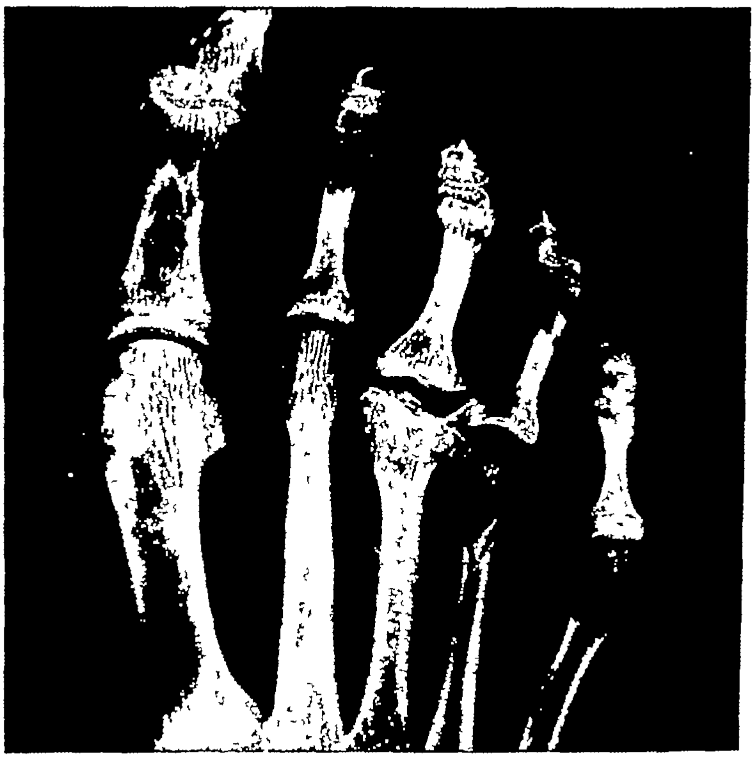


Fig 251 —Freiberg's infraction of third metatarsal head in a static state

Etiology.—The cause is not clear Freiberg (1914) at first stated that the disease was due to trauma When Kohler (1908) questioned this premise, he revised his opinion but left the question unanswered Panner (1921-1922) gave a detailed report of twenty-two cases of osteochondritis of the second metatarsal head, showing roentgenograms of various stages of the disease

Freiberg's infraction is currently (1959) believed to be due to an interference, by persistent pressure to the part, with circulation to the epiphysis during adolescence The predominance of the condition in the second metatarsal head lends support to this theory, because children are frequently fitted in short shoes, and the second toe, which is generally the longest of all the toes, presses against

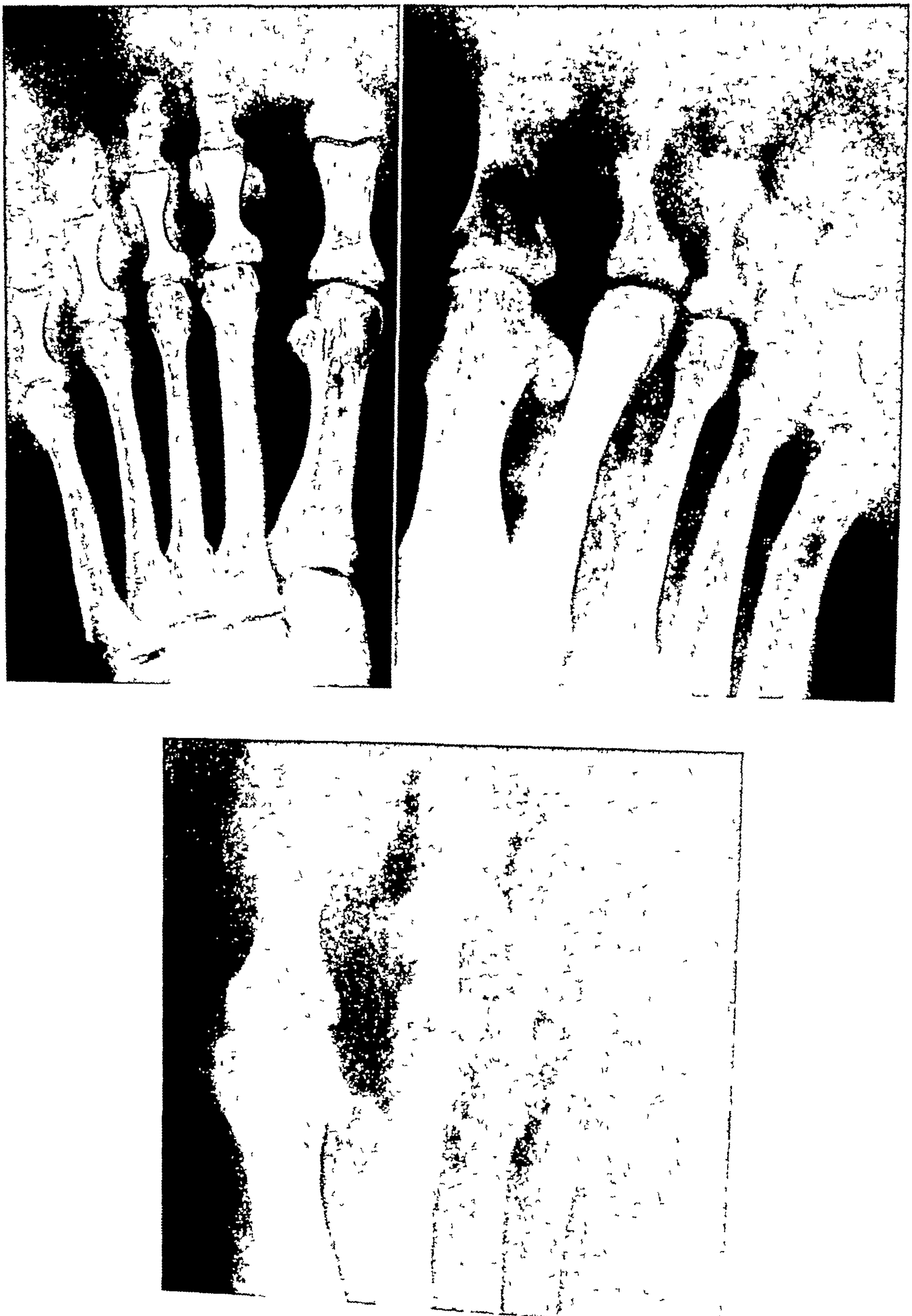


Fig 252 —Freiberg's infraction of second metatarsal head in active state, showing [illegible]

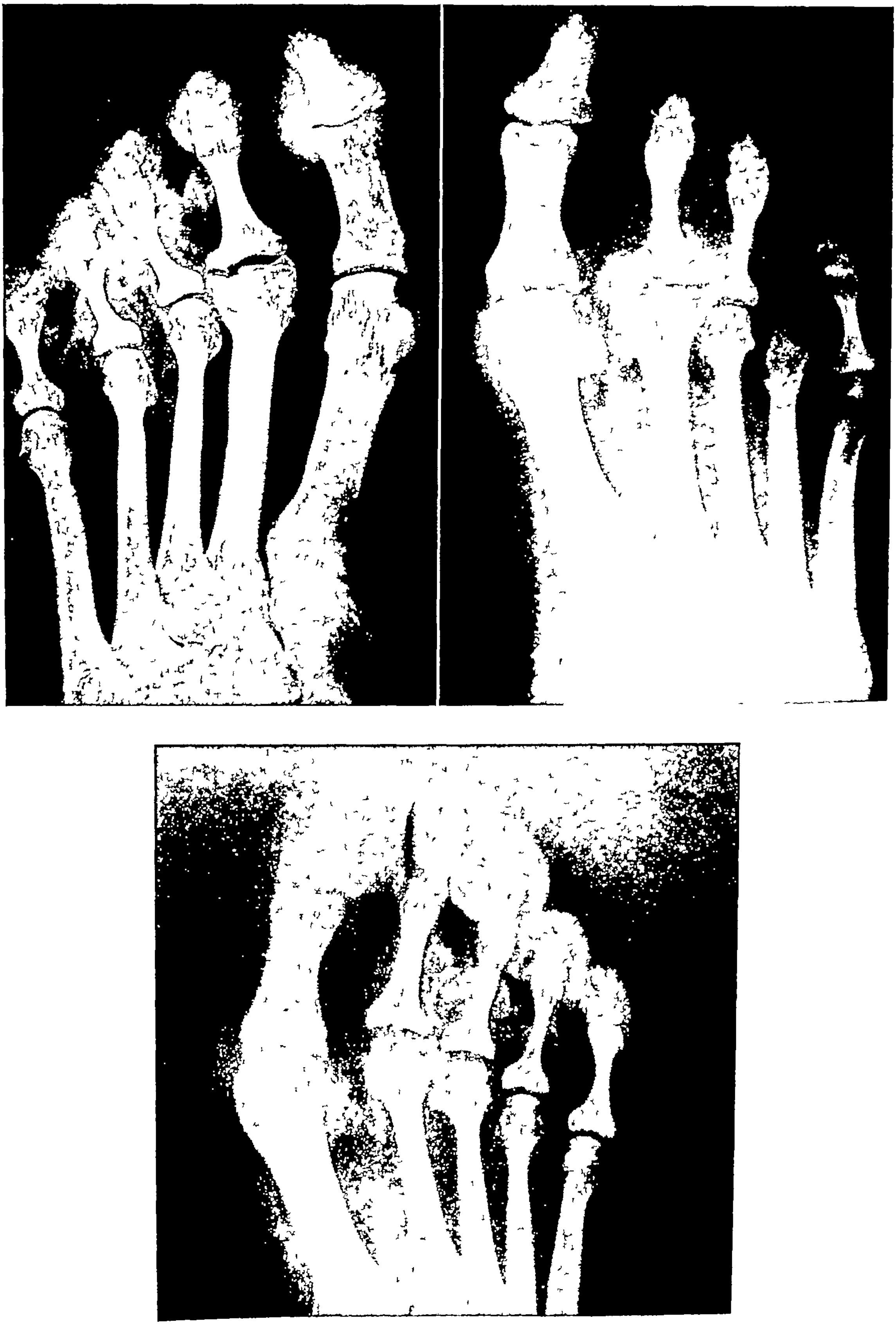


Fig 253 —Freiberg's infraction of second metatarsal head in static state

the boxing of the shoe until it is traumatized In certain children, this is undoubtedly the mechanism that triggers the injury to the epiphyseal plate of the metatarsal head sufficiently to cause necrosis of the second metatarsal head

Diagnosis.—The diagnosis is made roentgenologically, although the roentgenographic appearance varies according to the stage of the disease Watson-Jones (1952), Colonna (1950), and Van Demark and McCarthy (1946) warn that because the malady often produces periosteal thickening of the shaft of the second metatarsal, it may be mistaken for march fracture

Treatment.—In the acute stage, conservative and expectant measures are indicated When the degenerative process subsides, the condition becomes asymptomatic If the osteophytosis is extensive, it may become painful as a result of roughening in the metatarsophalangeal joint, or the sides of the head of the second metatarsal may be so enlarged as to grate against the adjacent metatarsal heads The enlargement may be on the plantar surface of the head, causing a painful weight-bearing area In such cases arthroplasty of the joint is indicated

Recommended Surgical Technique—Arthroplasty of the metatarsophalangeal joint is outlined

1 Make a dorsal incision over the metatarsophalangeal joint, extending from the web on either side of the head to the middle of the metatarsal shaft

2 Retract the skin margins and make a longitudinal incision in the capsule immediately over the metatarsophalangeal joint, retract the capsule margins with the skin margins

3 Make vertical incisions on each side of the capsule to free the head, so that it can be delivered dorsally (See page 188 for exposure of lesser metatarsal heads)

4 Excise all the excess bone and round the head with a rasp

5 Instill about 5 mg of hydrocortisone acetate into the joint

6 Suture the capsule and skin in layers and apply a compression bandage

Postoperative Care—The patient, wearing a cut-out shoe, may be ambulatory the second or third day In about three weeks a well-fitting shoe with a metatarsal support, such as an inlay or Thomas bar, should be prescribed

Kohler's Disease

Kohler's disease is an aseptic necrosis of the tarsal navicular occurring in children between the ages of 3 and 8 years The bone appears compressed anteroposteriorly and denser than normal It is usually unilateral but may be bilateral In Karp's study (1937) of forty-five patients treated at the Children's Hospital of Boston nine were bilateral cases O'Donoghue and his associates (1948) reported a bilateral case involving the navicular and first cuneiform Brailsford (1939) followed nine cases of osteochondritis of the tarsal navicular in adults which had the characteristic compression of the bone with gradual increase in density, however, the course was chronic and led to progressive deformity and a slipping or subluxating of the bone in most of his cases

Symptoms.—The symptoms are pain and swelling over the talonavicular joint Weight bearing may be tolerably or incapacitatingly painful On palpation or manipulation, the child tells of discomfort in the talonavicular joint

Diagnosis.—In the early stages, roentgenograms reveal the navicular to be osteoporotic and maldeveloped In advanced cases, the bone becomes sclerotic and compressed, and density (Fig 254) is greatly increased

Treatment.—Most cases are self-limiting and become asymptomatic in one to three years, finally regenerating sufficiently to leave a normal foot During the acute stage, conservative measures, such as limitation of function of the foot or complete immobilization by means of a walking cast, are indicated Surgical intervention should be resorted to only when such severe secondary changes take place as flattening of the head of the talus and formation of loose osteophytes on the articular surfaces In deforming and disabling cases, arthrodesis of the talonavicular and calcaneocuboid joints is required

Fig 254 —Osteochondritis of right navicular (Kohler's disease)

OSTEOCHONDRITIS OF CUBOID, SEVER'S DISEASE, TALUS INVOLVEMENT

Osteochondritis of Cuboid

It may be assumed that osteochondritis of the cuboid is rare because of the few publications Khoo (1950) reported such a case (Fig 255) Buchman (1933) and O'Donoghue and his associates (1948) reported cases of osteochondritis of the cuneiforms, but such reports are also few Treatment is the same as for osteochondritis of the navicular

Apophysitis of Calcaneus (Sever's Disease)

Apophysitis of the calcaneus was first described by Sever (1912), who reported five cases Thereafter little attention was given the disease Meyerding and Stuck (1934) found less than forty cases reported. They also analyzed twenty-one cases of apophysitis seen at the Mayo Clinic up to that time Simon and Williamson (1939) reported two cases

Mechanism.—The epiphysis of the heel is unique in being subject to both direct and indirect trauma. It is a *pressure epiphysis,* because of the weight-bearing function of the heel and the pressure exerted by the counter of the shoe The strong pull of the calf muscles inserted into it also makes it a *traction epiphysis* This predisposes the epiphysis to the combined causative factors of Legg-Calvé-Perthes disease, which is due to pressure, and Osgood-Schlatter's disease, which is due to traction *(osteochondrosis of the tuberosity of the tibia)*

Fig 255 —Osteochondritis of cuboid (From Khoo, F Y J Bone & Joint Surg 32B 230, May, 1950)

The calcaneal epiphysis is the only bone in the body whose epiphysis assumes the entire body weight before it is ossified, and it is the only one that has tremendous traction exerted on it without countertraction The strong traction is exerted by the calf muscles which are inserted into the epiphysis, no other muscles are inserted in the heel bone

Symptoms—The pathologic changes consist of an inflammatory process of the epiphysis, the epiphyseal plate, and the posterior portion of the body of the calcaneus The condition is usually unilateral but may be bilateral The onset

of symptoms is gradual, beginning as a slight pain at the back of the heel The patient walks with a limp and soon finds it uncomfortable to complete the step Pain may be experienced along the achillis tendon or in the calf muscles Children may walk on their toes in order to relax the pull of the achillis tendon

Examination and Diagnosis.—The disease occurs mostly in boys between the ages of 8 and 12 years, or during the period of their greatest growth An injury previously incurred may or may not be elicited in the history, although excessive participation in sports often is elicited The posterior part of the heel is tender on palpation, and there may be some thickening at the insertion of the achillis tendon Dorsiflexion of the foot is limited, the heel hurts on forced dorsiflexion

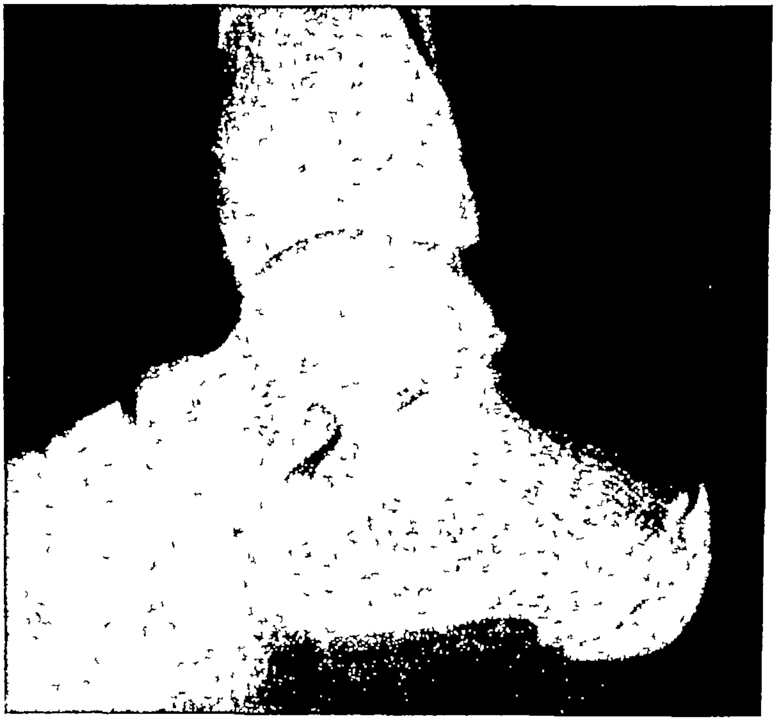


Fig 256 —Apophysitis of calcaneus Note increased density of epiphysis

The most convincing diagnostic feature is the characteristic roentgenographic changes In the lateral view of the foot the epiphysis of the heel may appear fluffy, motheaten, somewhat flattened, or partly fragmented, according to the stage of the disease (Fig 256) The adjacent posterior surface of the calcaneum is irregular Areas of rarefaction alternating with areas of increased density give this region a punched-out roentgenographic appearance Finally, the epiphyseal line appears cloudy and abnormally irregular

Treatment.—In mild cases, removal of the shoe counter, use of Gibney strapping and avoidance of excessive ankle function over a prolonged period relieve the symptoms In resistant cases, the condition may be alleviated by the application of a plaster cast with walking heel or caliper The cast should extend from the toes to just above the knee in such a manner as to hold the foot in slight equinus position and the knee in slight flexion That position relaxes the pull of the calf muscles The cast should be worn for six weeks or longer, after

which a well-fitting shoe may be worn The shoe may be wedged or contain an appliance to correct any coincidental foot imbalance The condition disappears at the age of 17 or 18 years, when the epiphysis becomes completely united

Osteochondritis of Talus

Osteochondritis of the talus is probably the most prevalent form of the disease, next to Freiberg's infraction, and is the most disabling form in the foot Until recently it had gone almost unnoticed As late as 1941 Mensor and Melody were able to find only twenty published cases, to which they added one case Since 1941, however, numerous contributions on the subject have appeared Aronsson (1942), Hutchison (1943), Ray and Coughlin (1947), Vaughan and Stapleton (1947), Marek (1949), Simpson (1950), Soeur (1950), Rodén and his co-workers (1953), Marks (1952), DeGinder (1955), and Cameron (1956), among others

Etiology.—It is accepted that most of the osteochondroses result from prolonged consistently neglected trauma This is true of osteochondritis of the talus Fairbank (1933), Mercer (1943), and Ray and Coughlin (1947) have supported this view

The medial trochlear surface of the body of the talus is affected most, because that part of the talus receives the greatest thrust and pressure from the body weight through the tibia Ray and Coughlin found necrosis on the mediosuperior aspect in eleven of fourteen of their patients In Rodén's series (1953) of fifty-five cases, forty were on the medial side.

Clinical Course.—The disease occurs during adolescence or in early adult life The onset is insidious and only sometimes is there a history of direct trauma The symptoms may point to the talonavicular joint and may simulate a strained longitudinal arch Pain and tenderness in the ankle joint may or may not be accompanied by swelling The swelling may be inflammatory or represent effusion into the joint Pain is aggravated by walking or exercising Partial immobilization with adhesive gives little or no relief Even a cast with a walking caliper may not completely alleviate pain It is partly relieved by rest and elevation Pain is the most important symptom, manifested in two phases (1) the active phase, when pain is due to an active inflammatory and degenerative process, (2) the static phase, when pain is due to the instability of the sequestrated ossicle which is loosely attached to the body of the talus (Fig 257)

Treatment.—During the active phase, conservative measures, such as limiting weight bearing and employing partial or complete immobilization, are indicated Asymptomatic cases discovered accidentally should be treated expectantly Those which become static and are asymptomatic do not need treatment but should be kept under observation

Recommended Surgical Technique.—Surgical intervention is indicated in static cases in which symptoms are distinctly referable to the site of the lesion and roentgenograms demonstrate complete separation of bone Because osteochondritis of the talus occurs mostly on its mediosuperior surface and because the medial

malleolus (Fig. 257) is in the direct path of this area, the approach to the lesion must be determined by its exact location in the longitudinal plane, which can be ascertained by a mediolateral roentgenogram of the ankle in extreme plantar flexion and a second view in extreme dorsiflexion. These roentgenograms indicate whether the area of necrosis will clear the articular surface of the tibia

If it clears completely on plantar flexion, which it ordinarily does, an anteromedial incision along the anterior margin of the medial malleolus permits the removal of the diseased bone with little difficulty, whereas, if it clears only in a dorsiflexed position, the bone may be removed through an incision behind and around the medial malleolus. This can be a difficult procedure, because the

Fig 257

Fig 258

Fig 257 —Osteochondritis of trochlear surface of talus Note loosely attached fragment on fibular side and the edema resulting from it

Fig 258 —Osteochondritis of trochlear surface of talus on tibial side Note complete separation of bone fragment

tendons of the tibialis posticus, flexor hallucis longus, and flexor digitorum longus, as well as the posterior tibial artery, vein, and nerve, are in the immediate path The more dorsiflexed the foot is, the more taut the tendons become, however, by careful dissection and blunt retraction, the tendon of the tibialis posterior can be retracted over the malleolus anteriorly and the tendons of the flexor hallucis longus and flexor digitorum longus with the posterior tibial artery, vein, and nerve retracted posteriorly, leaving sufficient space to remove the diseased bone

If the lesion is hidden from either approach, an osteotomy of the medial malleolus must be done to open the ankle joint The entire malleolus is removed

to expose the entire mediodorsal surface of the body of the talus and permit removal of the diseased bone The malleolus is replaced and fastened with a screw, and the ankle is held in mild inversion in a plaster splint for from nine to twelve days A walking caliper cast is worn for about six weeks.

When necrosis occurs on the laterosuperior aspect of the talus (Fig 258), the approach is simplified A vertical incision is made along the anterior margin of the lower end of the fibula, beginning about 3 cm above the superior surface of the talus, at the point over the sinus tarsi The incision is continued horizontally and anteriorly about 4 cm The lateral collateral ligaments are sectioned and retracted to permit the foot to be held in maximum adduction and inversion This exposes the laterosuperior surface of the talus The lesion is easily removed, because the circumscribed area of necrosis is detached from the body of the bone and is held in place by loose fibers The fibers can be freed with scissors The crater left by the removal of the bone is curetted out, the wound closed, and the foot immobilized.

ATROPHY OF BONE

OSTEOPOROSIS

Osteoporosis is a rarefaction or porousness of bone caused by a loss of bony substance. It is a symptom of several diseases. The disease may be general, such as Paget's disease or postmenopausal osteoporosis (Figs 259 and 260), or it may be local due to disuse, or, as is often encountered in the foot, due to Sudeck's atrophy

SUDECK'S ATROPHY (POSTTRAUMATIC ACUTE SUDECK-KIENBOCK'S BONE ATROPHY)

Sudeck (1900) first described the clinical entity referred to as *Sudeck's atrophy* He clearly differentiated the *acute* form from *chronic bone atrophy* that follows disuse Contributions to the subject have been numerous Cravener (1936), Gurd (1936), Hermann and Caldwell (1941), Jordan (1940), Reichle (1956) These are only a few who have reported their observations Sudeck's atrophy may occur in any part of the skeleton, but it has a predilection for the foot (Figs 261 and 262) The condition usually follows an injury to the foot but may follow an injury to the leg or thigh The injury may be minor or extensive Sudeck's atrophy may set in spontaneously, without a previous injury

Etiology.—There are several theories regarding the etiology of the disease. Sudeck regarded an inflammatory process as the important initial factor, whereas Schinz and his associates (1951) considered the primary factor a venous stasis, with engorgement in the haversian system which interferes with the return of tissue fluids into the blood stream This interference produces increased pressure inside the bone with resultant osteoclasis. Noble and Hauser (1926) thought that the disease is best explained on the basis of a reflex neurotrophic disturbance causing a reflex vasomotor spasm of the arterial blood supply Watson-Jones (1952) supported this view because, as he pointed out, the condition resembles causalgia from peripheral nerve injury Like causalgia, Sudeck's atrophy some-

Fig 259

Fig 260

Figs 259 and 260 —Postmenopausal osteoporosis Note generalized decalcification

Fig 261

A B

Fig 262

Figs 261 and 262 —Sudeck's atrophy *A*, Normal foot for comparison *B*, Note generalized decalcification and mottling of bones

times responds favorably to sympathetic block. This suggests that afferent impulses from the region of injury cause a reflex neurovascular sympathetic disorder in the foot

Pathogenesis.—The loss of bone substance involves the entire foot Loss is greater in the spongiosa, somewhat less in compact bone Decalcification predisposes the person to spontaneous fracture, especially of the metatarsals (Fig 263) Osteoporosis is readily demonstrable on roentgenograms taken of both feet

Clinical Diagnosis.—The soft tissue changes are a static edema, redness of the skin, and generalized pain in the entire foot The history falls into three stages (1) the initial injury, which may be minor, (2) the latent or symptomless period, which may last one or two months, and (3) the acute stage, which

Fig 263—Sudeck's atrophy of left foot with march fracture of second metatarsal, resulting from softening of bone

begins with pain, swelling, and redness of the entire foot, reaching greatest severity in two or three days The symptoms are aggravated by weight bearing

The disease is typically unilateral, although spontaneous bilateral involvement has been reported Roentgenologically, the affected foot shows extensive osteoporosis and mottling of some or all of the bones of the foot, demonstrated by comparison with the normal foot

Treatment.—Many different types of medication have been tried in the treatment of Sudeck's syndrome Almost in every case medication has been combined with other methods of treatment Little practical improvement through medication has ever been reported Complete immobilization for about four weeks followed by partial immobilization to prevent further trauma is the only accepted form of therapy

Prognosis.—The disease is self-limiting, the acute stage lasts from two to four months, followed by a chronic and reparative stage lasting about a year.

OSTEOSCLEROSIS

Osteosclerosis (marble-bone disease) is a condition wherein the bones increase in density, with varying degrees of obliteration of the medullary cavity. Locally, the condition may follow chronic osteomyelitis, degenerative arthritis, or abnormal mechanical stress on a given part of the skeleton. A generalized form of osteosclerosis is sometimes encountered, known as *Albers-Schonberg disease* Luck (1950) considered the term *marble bone* erroneous, because the bone is more like chalk than marble.

A diagnosis is made roentgenologically by the observation of massive increase in the density of the bones, with partial or complete obliteration of the medullary canal The disease may be accompanied by various deformities of the extremities which are only rarely amenable to attempts at surgical correction.

ARTHRITIDES

Because the joints of the foot are so frequently the site of local trauma, they are inevitably subject to traumatic arthritis The foot is likewise the site of generalized arthritides The fundamentals and complexities of the diseases that are destructive of the joints of the foot are not within the coverage of this text Some examples, necessary to intended inclusiveness and to arouse the student's awareness, are presented herewith

GOUT

Gout (podagra) is a disease characterized by a disturbance in purin metabolism which results in an increase in uric acid in the blood, above 5 mg per cent, and also in deposition of sodium biurate salts in various parts of the body, mainly in the periarticular structures

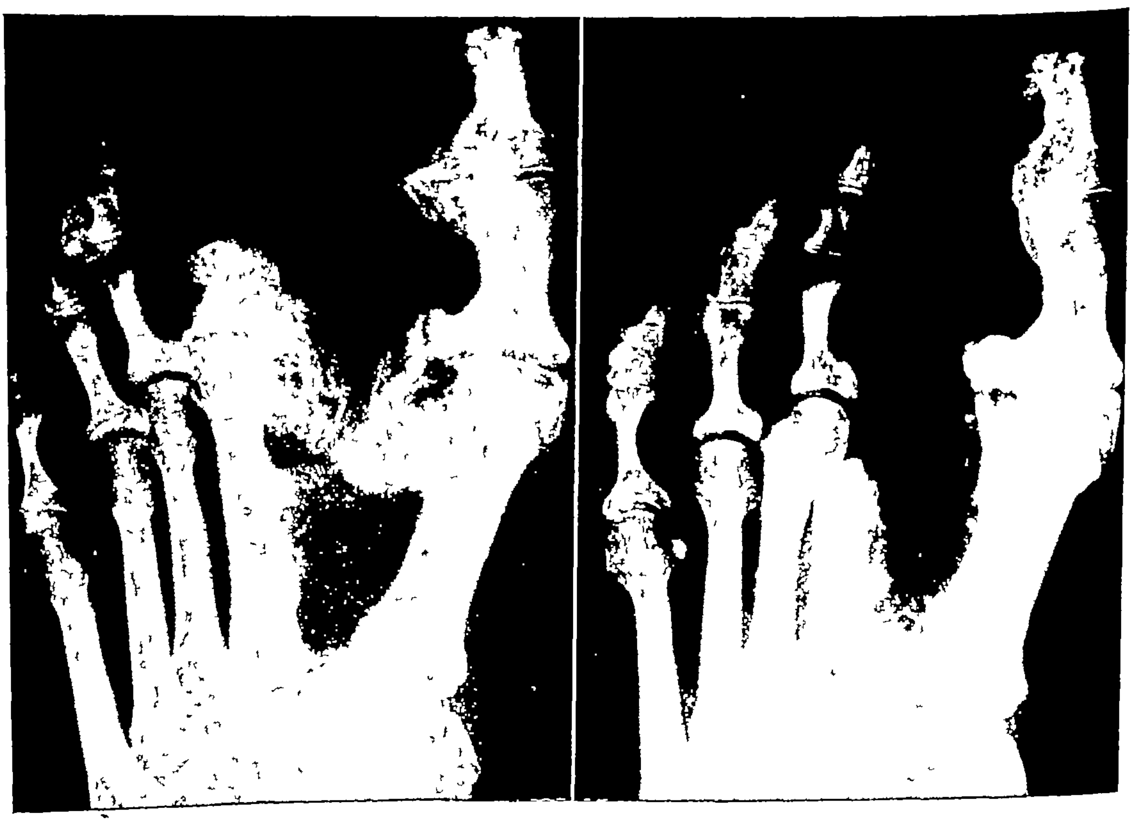
The symptoms are recurrent attacks of acute arthritis, especially in the first metatarsophalangeal joint The onset of an attack is sudden The affected joint is swollen, tender, and extremely painful An acute attack subsides in a few days but recurs at intervals The articular surface and the area around it undergoes degenerative changes (Fig 264)

TRAUMATIC ARTHRITIS

Sudden or prolonged injury to the joints of the foot are common, because of its weight-bearing function and as a result of ill-fitting shoes. Contributing factors to injury of the joints of the foot are prolonged standing and walking on hard surfaces, poor gait, and poor posture Anatomic deviations may increase pressure and irritation on one or more joints The joint most traumatized on this account is the first metatarsophalangeal joint (hallux rigidus, page 392) (Figs 265 to 268)

Fig 264 —Gouty arthritis of first, second, and third metatarsophalangeal joints Note punched-out areas

A



B

Fig 265 —*A*, Traumatic arthritis, causing bizarre bone reaction *B*, After removal of new bone formation

DEGENERATIVE ARTHRITIS

Osteoarthritis, senile arthritis, and *hypertrophic arthritis* are interchangeable terms for degenerative arthritis of aging Many conditions accelerate the process of this type of arthritis, for example, injury, either a severe single episode or repeated mild injuries The osteoarthritic end result may not become symptomatic until the climacteric. Changes in the joints consist of diminishing cartilage from compression and wear and increased density of the subchondral bone (Fig 269) Clubbing of the terminal joints of the fingers (Heberden's nodes) is also characteristic

Fig 266

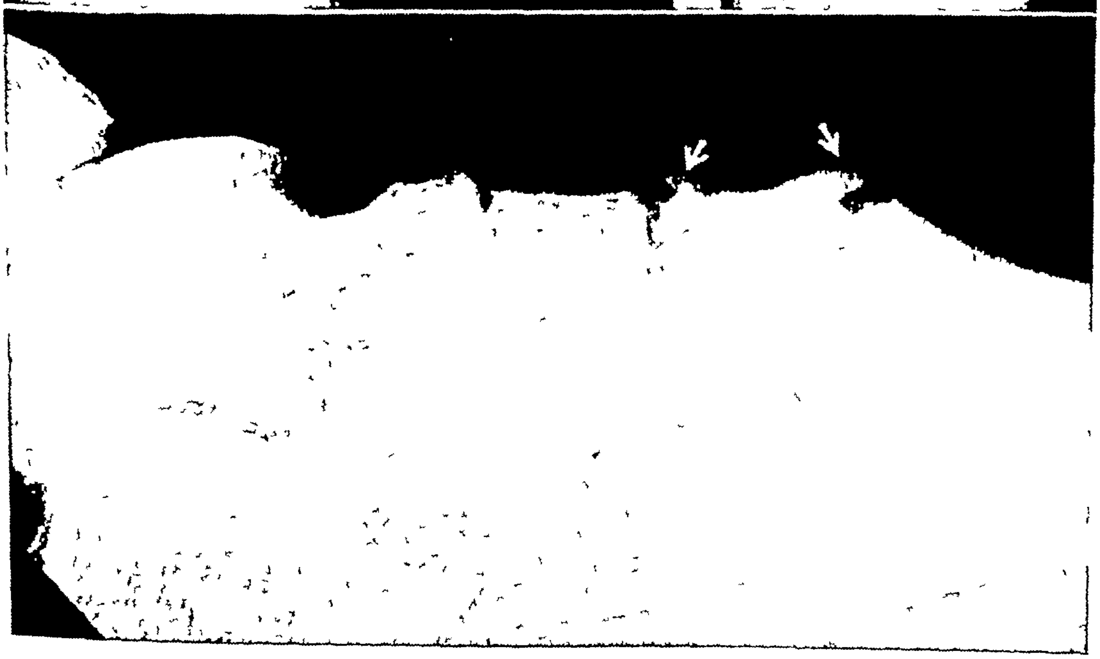


Fig 267

Fig 266 —Traumatic arthritis of first metatarsophalangeal joint (hallux rigidus), showing extensive reactive bone proliferation

Fig 267 —Traumatic arthritis of first metatarsal-first cuneiform articulation

A B.

Fig 268 —*A* and *B*, Traumatic arthritis of both first metatarsophalangeal joints *A*, Note compensatory reaction of third metatarsal and extensive osteosclerosis

Fig 269 —Osteoarthritis of talotibial articulation Note complete absence of cartilage and compression of subchondral bone

Fig 270 —Rheumatoid arthritis of first four metatarsophalangeal joints Note atrophic changes of articular surfaces of these joints

Fig 271 —Rheumatoid arthritis Typical deformity of all metatarsophalangeal articulations

Fig 272 —Rheumatoid arthritis in a skeleton specimen

Fig 273 —Complete arthrodesis of talotibial articulation consequent to acute septic arthritis of this joint

RHEUMATOID ARTHRITIS

Rheumatoid arthritis is also called *atrophic arthritis, arthritis deformans, polyarthritis,* or *nonspecific infectious arthritis* This disease has many of the characteristics of an infectious process It is sometimes classified among the collagen diseases The joint changes are diffuse decalcification, atrophy of bone and of cartilage, atrophy of interossei muscles, marginal erosion, and synovial distention The feet become characteristically deformed as a result of degeneration of the metatarsophalangeal joint (Figs 270 to 273) and atrophy of the interossei muscles

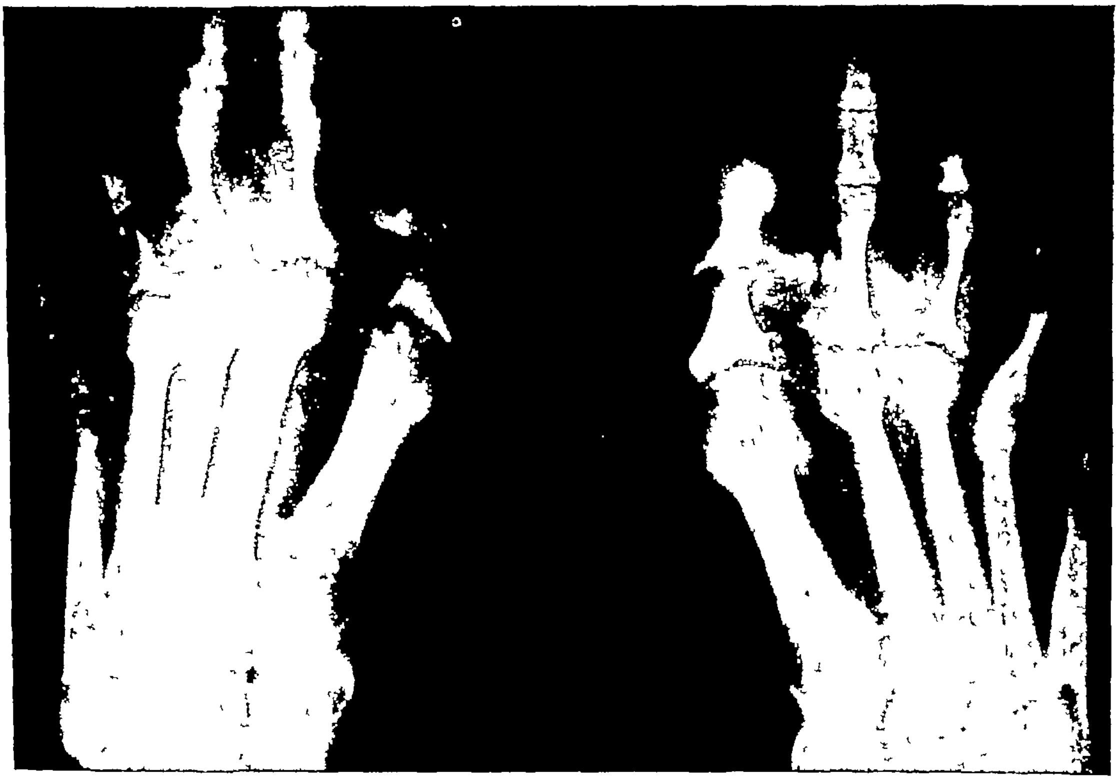


Fig 274 —Psoriatic arthritis of both feet Note extensive destruction of some of proximal phalanges

PSORIATIC ARTHRITIS

Psoriatic arthritis is a form of arthritis observed at times in cases of longstanding psoriasis, in which there are episodes of acute exacerbation of joint symptoms The joints of the feet are often involved (Fig 274) The changes may vary from an inflammation of the synovia to complete atrophic destruction of the joint and adjacent bone It has been suggested that this phase of psoriasis is closely related to rheumatoid arthritis

REFERENCES

Arkin, A M, and Schein, A J Aseptic Necrosis in Gaucher's Disease, J Bone & Joint Surg 30A 631-641, July, 1948

Aronsson, H Osteochondritis of the Ankle Joint, Zentralbl f Chir 69 312, Feb 21, 1942

Brailsford, J F Osteochondritis of the Adult Tarsal Navicular, J Bone & Joint Surg 21 111, Jan, 1939

Breitenfelder, Heinrich· Gibt es eine dem Morbus Kohler II analoge Affektion auch am Kopfchen des Os Metatarsale I, Ztschr Orthop 46 181, 1937
Buchman, Joseph Osteochondritis of Internal Cuneiform, J. Bone & Joint Surg 15 225-232, Jan, 1933
Cameron, B M Osteochondritis Dissecans of the Ankle Joint, J Bone & Joint Surg 38A 857-861, July, 1956
Colonna, P C Regional Orthopedic Surgery, Philadelphia, 1950, W B Saunders Co, p 482
Cravener, E K Acute "Reflex" Bone Atrophy (Sudeck's Disease), New York J Med 36 815, May 15, 1936
DeGinder, W L Osteochondritis Dissecans of Talus, Radiology 65 590-598, Oct, 1955
Fairbank, H A T Osteochondritis Dissecans, Brit J Surg 21 67-82, 1933
Freiberg, A H Infraction of Second Metatarsal Bone, Surg. Gynec & Obst 19 191, July, 1914
Freiberg, A H So-called Infraction of Second Metatarsal Bone, J Bone & Joint Surg 8 257-261, April, 1926
Gurd, F B Posttraumatic Acute Bone Atrophy, A Clinical Entity, Arch Surg 32 273, Feb, 1936
Haglund, Patrik Ueber Fractur des Epiphysenkerns des Calcaneus, nebst allgemeinen Bemerkungen uber einige ahnliche juvenile Knockenkernverletzungen, Arch f klin Chir 82 922-930, 1907
Hermann, L G, and Caldwell, J S Diagnosis and Treatment of Posttraumatic Osteroporosis, Am J Surg 51 630, March, 1941
Hutchison, R G Osteochondritis Dissecans, Brit J Radiol 16 147-149, May, 1943
Jordan, H H After Care of Fractures With Special Reference to Delayed Union and Sudeck's Atrophy, Arch phys Therap 21 25, Jan, 1940
Karp, M F Kohler's Disease of the Tarsal Scaphoid, J Bone & Joint Surg 19 84, Jan, 1937
Khoo, F Y Osteochondritis of Cuboid Associated With Tuberculosis of Adjacent Tarsal Bones, J Bone & Joint Surg 32B 230, May, 1950
Kohler, Alban Ueber eine haufige, bisher anscheinend unbekannte Erkrankung einzelner kindlicher Knochen, Munchen med Wchnschr 55 1923, 1908
Luck, J V Bone and Joint Diseases, Springfield, Ill, 1950, Charles C Thomas, Publisher, p 36
Marek, F M Osteochondritis Dissecans of the Talus in Recurrent Ankle Sprains, New York J Med 49 163, Jan 15, 1949
Marks, K L Flake Fracture of Talus Progressing to Osteochondritis Dissecans, J Bone & Joint Surg 34B 90, Feb, 1952
Mensor, M C, and Melody, G F Osteochondritis Dissecans of Ankle Joint, Use of Tomography as a Diagnostic Aid, J Bone & Joint Surg 23 903-909, Oct, 1941
Mercer, W Orthopaedic Surgery, ed 3, Baltimore, 1943, Williams & Wilkins Co
Meyerding, H W, and Stuck, W G Painful Heel Among Children, J A M A 102 1658, May 19, 1934
Mouchet, A Metatarsal Epiphysitis, J Bone & Joint Surg 11 87-93, Jan, 1929
Noble, T P, and Hauser, E D W Acute Bone Atrophy, Arch Surg 12 75-94, pt. 1, Jan, 1926
O'Donoghue, A F, Donohue, E S, and Zimmerman, W W Bilateral Osteochondritis of the Tarsal Navicular and the First Cuneiform, J Bone & Joint Surg 30A 780, July, 1948
Osgood, R B Lesions of the Tibial Tubercles Occurring During Adolescence, Boston M & S J 148 1903
Panner, H J A Peculiar Characteristic Metatarsal Disease, Acta radiol 1 319-333, 1921-1922
Ray, R B, and Coughlin, E J Osteochondritis Dissecans of Talus, J Bone & Joint Surg 29 697, July, 1947
Reichle, R Das Sudeck-Syndrom, Langenbecks Arch klin Chir 284 18, 1956
Rodén, Sture, Tillegard, Paul, and Unander-Scharin, Lars Osteochondritis Dissecans and Similar Lesions of Talus A Report of 55 Cases With Special Reference to Etiology and Treatment, Acta orthop scandinav 23 51, 1953 (abstracted, Surg Gynec & Obst 98:5, May, 1954)
Schaefer, R L, Strickroot, F L, and Purcell, F H Endocrine Implication of Juvenile Chondro-Epiphysitis, J A M A 112 1917-1919, May 13, 1939
Schinz, H R, Baensch, W E, Friedl, E, and Uehlinger, E Roentgen-Diagnostics, vol 1, New York, 1951, Grune & Stratton, Inc
Schlatter, C Meniscusluxationen, Beitr z klin chir 41 229-245, 1904
Sever, J W Apophysitis of the Os Calcis, New York J Med 95 1025, 1912
Shaw, E W Avascular Necrosis of Phalanges of Hands (Thiemann's Disease), J A M A 156 711-713, Oct 16, 1954

Simon, H E, and Williamson, B Epiphysitis of the Os Calcis, J Bone & Joint Surg 21 1015, Oct, 1939
Simpson, T R A Case of Osteochondritis Dissecans of Ankle, Brit J Surg 37 359-360, Jan, 1950
Soeur, R Osteochondritis Dissecans of Ankle, Acta orthop belg 16 493, 1950 (abstrated, Internat Abstr Surg Gynec & Obst, p 578, 1954)
Sudeck, Paul· Ueber die acute entzundliche Knochenatrophie, Arch klin Chir 62 147, 1900
Van Demark, R E, and McCarthy, P V Panner's Metatarsal Disease A Condition of Aseptic Necrosis Simulating March Fracture, J Bone & Joint Surg 28 842, Oct, 1946
Vaughan, C E, and Stapleton, J G Osteochondritis Dissecans of Ankle, Radiology 49 72, July, 1947
Watson-Jones, Reginald Fractures and Joint Injuries, ed 4, Baltimore, 1952, Williams & Wilkins Co, vol 1, pp 53, 347.

CHAPTER FIFTEEN

Fractures and Dislocations

FRACTURES OF BONES OF THE FOOT (EXCLUDING MAJOR FRACTURES OF CALCANEUS AND TALUS)

A FRACTURE IS A BREAK, OR RUPTURE, IN THE STRUCTURAL INTEGRITY OF BONE OR cartilage.

Predisposing Causes

In children, incomplete ossification increases the likelihood of epiphyseal and avulsion fractures. In the elderly, senile bone atrophy, or senile osteoporosis, predisposes to fractures. Young adults and the middle aged, especially laborers and athletes, are subjected to hazards of occupation and activity causing accidental fractures. General bone fragility may be a consequence of malfunction of the parathyroids or a symptom of rickets or osteomalacia. Local diseases of bone, such as deformity, bone cysts, or localized necrosis, increase susceptibility to fracture.

Immediate Cause

Direct violence is ordinarily causative, but the cause may be indirect, for example, in bracing oneself during an accident, a bone anywhere in the body may break. Sudden spasmodic contraction of a muscle or a group of muscles may cause a fracture in such areas as the patella or the calcaneus (avulsion fracture).

Types of Fracture

Closed, or *simple,* fractures are those that do not connect with an open wound; *open,* or *compound,* fractures do have such communication. An *incomplete* fracture, or *infraction,* in an adult means simply that the bone was not

completely broken; in children, an incomplete fracture is known as a *greenstick* fracture in which, because children's bones are flexible, one side of a bone is broken but the opposite side is bent *Complete* fractures may be transverse from direct violence, oblique from indirect violence, or spiral from rotation or twisting, but regardless of the direction, the fracture line extends completely across. A crushed or fragmented bone is called a *comminuted* fracture (Fig 275) In an *impacted* fracture, one segment of bone is driven into another. In a *complicated* fracture, muscles, nerves, or blood vessels are torn or entangled at the fractured site

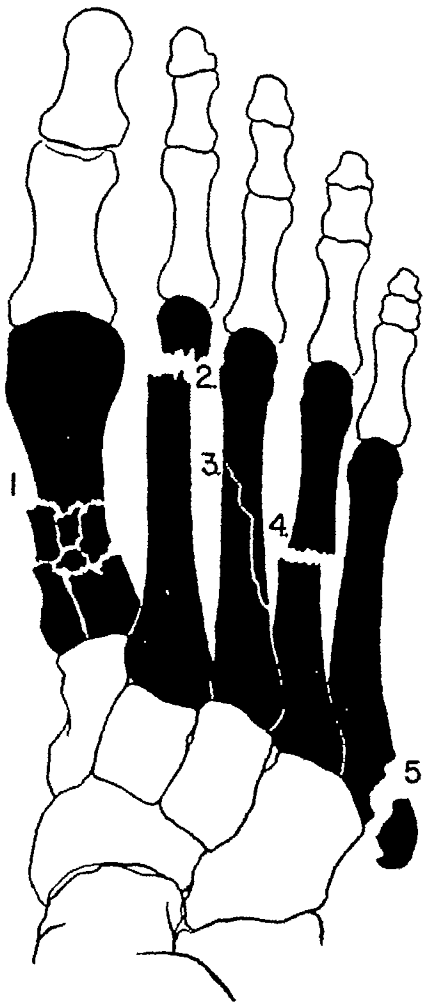


Fig 275 –Types of fractures *1*, comminuted, *2*, transverse with displacement, *3*, oblique or spiral, *4*, transverse, and *5*, avulsion fracture of tuberosity (After Netter)

Healing of Fractures

The healing of fractures is a continuous process but may be thought of as a four-stage progression (1) During the first week a blood clot forms around the ends of the fractured bone and beneath the periosteum, the clot congeals into a gelatinous mass (2) During the second week the fractured site becomes fixed in granulation tissue, which replaces the clot Young bone cells begin to be laid down around the vessels, the medullary tissue is absorbed and replaced by

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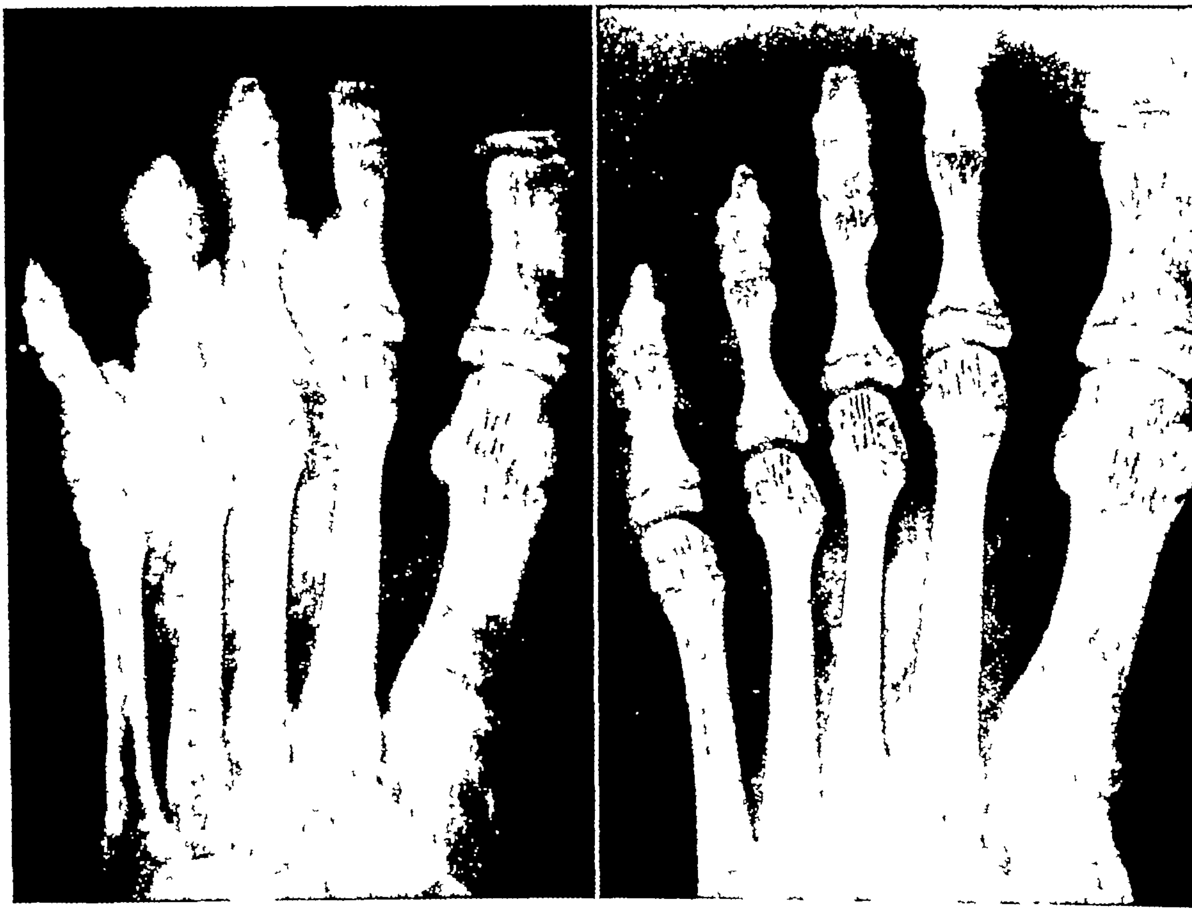


A B

Fig 276—A, Fracture-dislocation at epiphyseal plate at base of fifth proximal phalanx B, After closed reduction and healing

around the metatarsophalangeal joints limit motion in those joints Immobilization is maintained for about four weeks Ambulation is permitted immediately, provided the sole of the shoe is semirigid

Hallux Phalanges

Fracture of the hallux phalanges presents a more difficult problem than fracture of the other phalanges The bones are comparatively large, and their function is as important as all the other phalanges together The proximal phalanx of the hallux is the one broken most frequently (Fig 278)

If there is no displacement, the hallux should be bound to the two adjacent toes and a walking cast extending beyond the toes applied and maintained for four weeks When displacement is present but cannot be reduced by manipulation, open reduction is indicated (Fig 279)

Technique for open reduction of fractured first proximal phalanx is herewith outlined

1 Make a 6 cm longitudinal incision over the hallux, extending from behind the eponychium proximally

2 Incise the capsule and retract the margins, including the extensor longus hallucis tendon

callus (3) During the third week the conversion of callus into bone begins to take place The callus becomes denser through calcification, especially around its blood vessels, the original bone rarefies as its vessels become continuous with those of the callus (4) In the final stage, which takes place in from four to eight weeks, the external and internal callus disappears and the intermediate callus condenses into hard bone In adults, completion of the healing process may take as long as a year

Principles of Treatment of Fractures

The objectives of treatment of any fracture are (1) to secure union of the broken fragments, (2) to obtain normal function of the area, (3) to obtain ideal results at the earliest possible time All methods of treatment must be weighed against the foregoing considerations, as well as in terms of the welfare of the patient and respect for tissues As in all surgical problems, the surgeon must never forget that all manipulative and operative procedures injure tissue, the ideal operation traumatizes as little as possible while attempting to eradicate existing disease

Regarding both open and closed reduction of a fracture, roentgenograms should be taken immediately after attempted reduction in order to ascertain whether reduction has been accomplished Roentgenograms are taken repeatedly at frequent intervals to ascertain whether reduction has been maintained

Immobilization of fractures with circular casts always risks the possibility of constricting swelling by the rigidity of the cast Constriction can obstruct the arterial flow and lead to gangrene That is why casts, especially those applied shortly after injury, should be well padded, especially over bony prominences Frequent examinations for signs of ischemia are necessary

FRACTURES OF THE PHALANGES

Fifth Proximal Phalanx

The commonest fracture of the forefoot is of the fifth proximal phalanx Stubbing the fifth toe is the usual cause Among children, the fracture may be at the epiphyseal plate (Fig 276), among adults, the fracture is likely to be of the shaft (Fig 277)

Phalanges of Middle Toes

Fracture of any of the three middle toes is uncommon, when it does occur, it is the proximal phalanx, a relatively long bone, which is broken The middle and distal phalanges are seldom fractured

Displacement is rare in fracture of the phalanges of the three middle toes, when it does occur, it can be readily reduced, often without an anesthetic

The adjacent toes may serve as a splint Adhesive tape, ½ inch wide, is wound around the fractured toe as well as around the two or three adjacent toes, so that none of the toes can move independently Strips of adhesive wound

C

Fig 277 (cont'd) —For legend see opposite page

Fig 278 —Impacted fracture of proximal phalanx of hallux

3 Force the segments of bone into position with a small blunt bone elevator

4 It may be necessary to use a pin or small staple (Fig 279) to maintain fixation, if so, it should be removed in about six weeks

5. The patient wears a walking cast for four or five weeks The cast should extend beyond the toes

Sesamoids

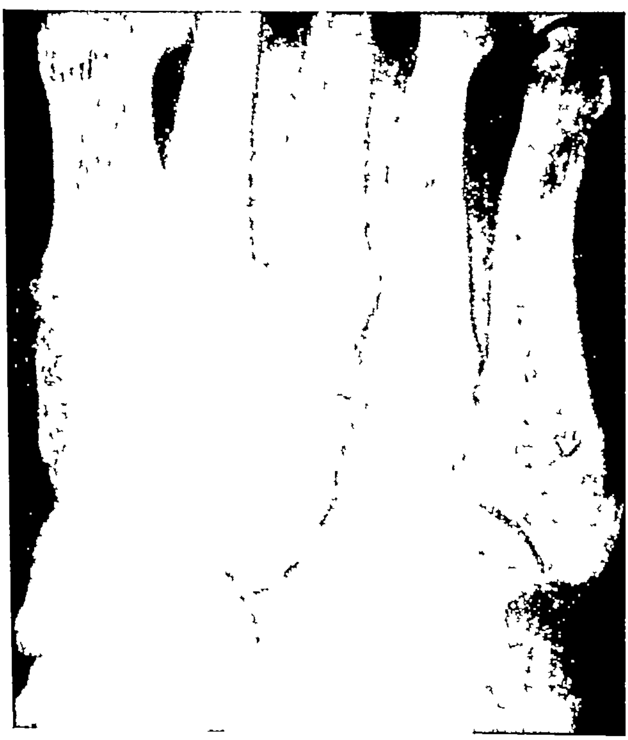
Fracture of the sesamoids is discussed separately in Chapter 12, Disorders of the Sesamoids

A

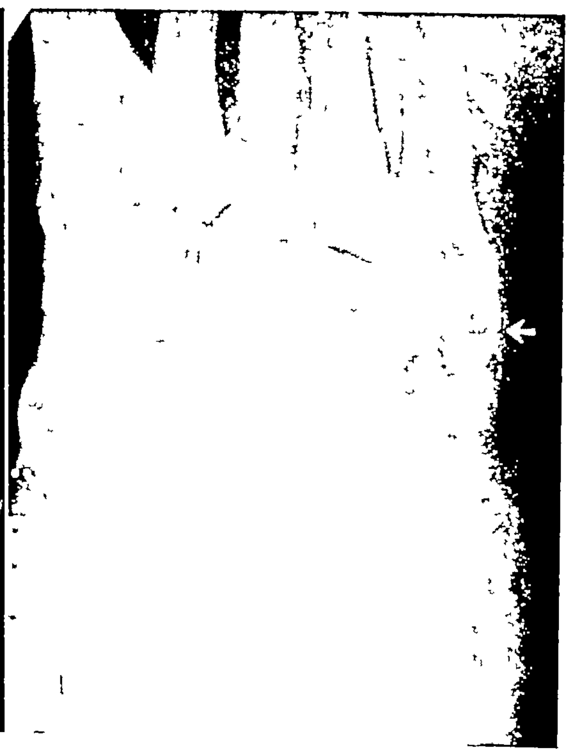
B

Fig 277 —*A*, Fracture at base of fifth proximal phalanx *B*, Fracture at neck of fifth proximal phalanx Neglect resulted in nonunion *C*, Fracture of shaft of fifth proximal phalanx

A.



B



C

Fig 280 —*A*, Transverse fracture at proximal end of fifth metatarsal *B* and *C*, Infraction at base of fifth metatarsal *D* and *E*, Fracture of tip of base of fifth metatarsal

(*Fig 280 continued on next page*)

A

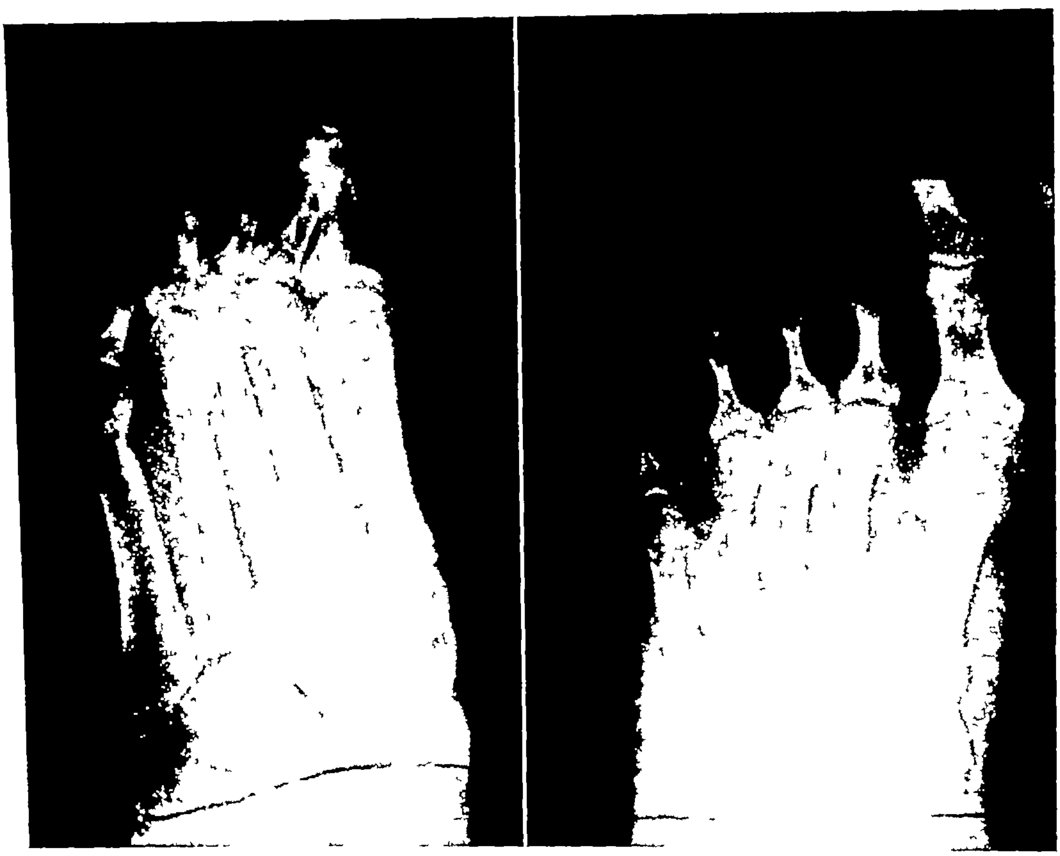
B

Fig 279 — *A*, Impacted fracture and displacement of proximal phalanx of hallux *B*, After open reduction and fixation, staple removed six weeks postoperatively

FRACTURES OF METATARSAL BONES

The *base of the fifth metatarsal bone* is a frequent site of fracture (Fig 280) which can produce complications because the peroneus brevis inserts into it and the peroneus longus tendon is held by it in the cuboid groove Among children, fracture of the base of the fifth metatarsal bone must be differentiated from the epiphyseal line Ordinarily there is no displacement

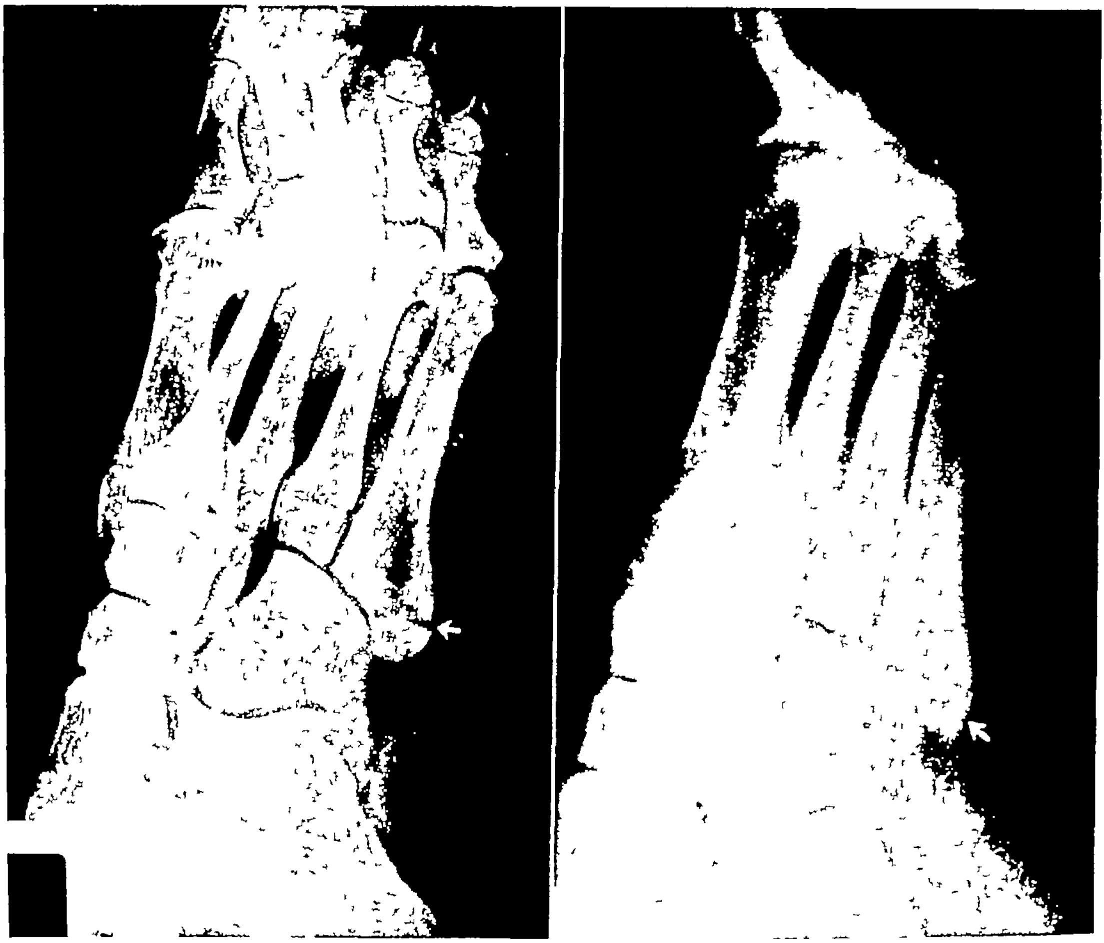
A.



B

Fig 281 —*A*, Fracture of neck of third and fourth metatarsals *B*, Fracture of neck of fourth and fifth metatarsals *C*, Ununited fracture of head of third metatarsal *D*, Oblique fracture of fifth metatarsal shaft *E*, Malunited fracture of fifth metatarsal shaft

(*Fig 281 continued on next page*)



D E

Fig 280 (cont'd) —For legend see page 325

Strapping the foot is the only treatment required in most instances Strapping is done by binding the base and heads of all the metatarsals with adhesive Sometimes a walking cast is needed for about six weeks In cases of severe avulsion of the broken fragment, open reduction may be required in which the fragment is pinned through the shaft and maintained while the patient wears a walking cast for six weeks

Simple fracture of the neck or shaft of the lesser metatarsal bones is likely to take place spontaneously Fractures of the lesser metatarsal bones resulting from injury are, for the most part, multiple (Fig 281) and usually occur at the neck, which is the weakest part of the metatarsal bones Such fractures may be serious when displacement of the heads is extensive If displacement is plantarwise and not reduced, disability may result

Extensive injury to the plantar arterial arch and to the dorsalis pedis may accompany a fracture and extreme displacement of the proximal part of the lesser metatarsals Gissane (1951) reported three cases of Lisfranc's fracture with gross displacement which were treated conservatively but ended in below-the-knee amputation, because of impairment of blood supply to the foot through "twisting of the main vessels" Because of this real danger, Gissane prefers open

A B

Fig 282—A, Oblique fracture of fifth metatarsal shaft, with displacement Blood supply may be affected B, After open reduction and fixation with stainless steel wire

A B

Fig 283—A, Neglected fracture at base of first metatarsal, concomitant fracture of neck of second metatarsal B, Old fracture of first and second metatarsals

C *D*

E

Fig 281 (cont'd) —For legend see page 327

5 Withdraw the wire "through the skin until the blunt end is level with the fracture site"

6 Appose fragment ends, drill "proximally until blunt end of wire meets resistance at the base of the metatarsal bone."

7 Fit a small cork over distal (exposed) end of the wire for protection, and immobilize the limb in plaster for five weeks

8 After removal of the cast, remove the wire. Removal of the wire is readily accomplished at this time

FRACTURES OF MIDTARSAL BONES

Fracture of the cuboid is not common but not so rare as reports indicate Hermel and Gershon-Cohen (1953) reported five cases They found that the condition was not mentioned in most textbooks and seldom referred to at all in any publications during the previous twenty years I have seen three such cases (Fig 285) Fracture is usually by compression, there is slight displacement The site is at the plantar aspect of the bone, where it is wedged in between the head of the calcaneus and the base of the fifth metatarsal bone Its painfulness is due to irritation that the fracture sets up in the peroneus longus tendon which glides under the cuboid

Three personal patients responded to conservative measures of immobilization for five weeks in walking casts Among the cases reported by Hermel and Gershon-Cohen, one patient was treated by midtarsal fusion because of severe comminution It is conceivable that a fracture of the cuboid may require major measures, but this would not happen often

Fracture of the cuneiforms without disruption of the entire tarsometatarsal ensemble is rare, when it happens, the medial cuneiform is the one likely to be affected Holstein and Joldersma (1950) reported two such cases

Reduction of fracture-dislocation of the medial cuneiform is by countertraction and manipulation Traction may require inserting one pin through the distal third of the metatarsal bones and one through the body of the calcaneus The pins are incorporated in the cast Sometimes open reduction and stabilization are necessary

Tarsometatarsal fracture-dislocation (Fig 286) is not so uncommon as is sometimes believed Easton (1938) spoke of it as a rare dislocation at Lisfranc's joint, but Manuel del Sel (1955) encountered thirteen cases within eighteen months

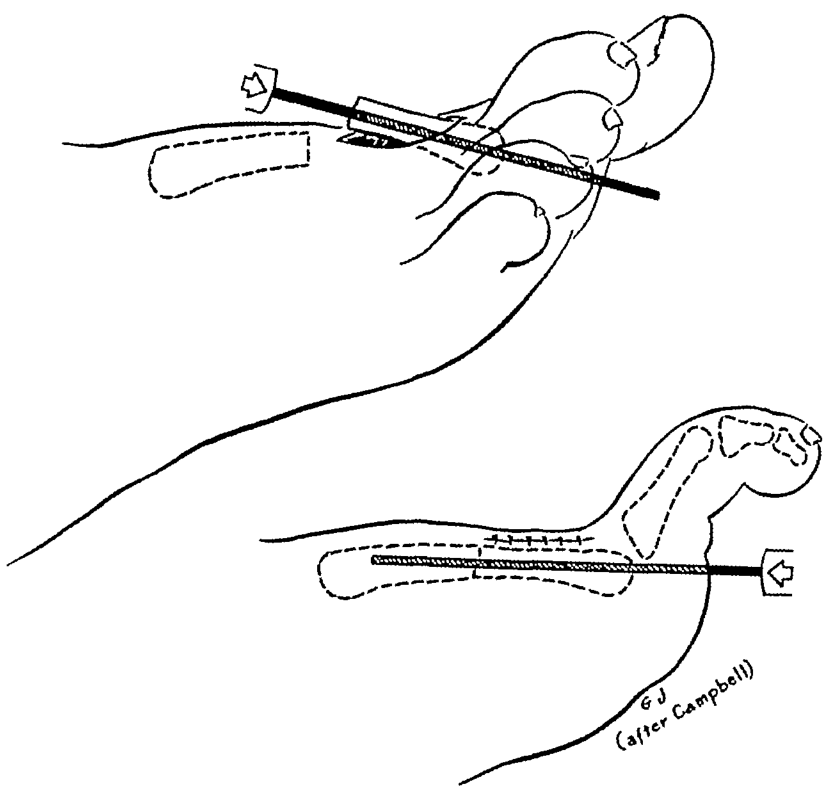
Most tarsometatarsal dislocations can be reduced by manual traction and manipulation, especially if done promptly Immobilization by a well-padded plaster cast, elevation of the leg for a week, and refraining from bearing weight for six to eight weeks will complete the treatment successfully As Gissane has pointed out, however, this can be a serious type of fracture The abducting force that dislocates the forefoot may tear the arteries in the foot and twist and stretch the vessels and nerves behind the medial malleolus so as to threaten gangrene or paralysis of the foot

reduction. Certainly the status of the blood supply must always be evaluated before any treatment is instituted, however, it is usually possible to reduce the displacement by manipulation and the wearing for six weeks of a walking cast that includes the toes.

Traction by means of loops of wire passed through the toes and fixed to a banjo splint has been advised, but its value is doubtful. At times, when manipulative reduction fails, it may be necessary to correct the displacement by open reduction and the use of pins or wire (Fig. 282) to maintain apposition.

Fracture of the first metatarsal bone is rare because of the strength of this bone. When the bone does fracture, displacement is slight (Fig. 283), immo-

A



B

Fig. 284.—*A*, Steinmann pin passed through distal end of fractured metatarsal. *B*, Pin guided into shaft of proximal end of metatarsal. (After Campbell.)

bilization for six weeks suffices. Displacement can be reduced by closed reduction and, if necessary, maintained by a pin (Fig. 284) as advised by Campbell (1956).

1. Expose the metatarsal fractures through a small dorsal incision.
2. Lift out the proximal end of the distal fragment.
3. Drill the pointed end of a small Kirschner wire distally into medullary part of the distal fragment while the toes are held in dorsiflexion. Drill until the point of the wire emerges.
4. Remove the drill from the wire and reverse it.

Fig 286 —Tarsometatarsal fracture-dislocation

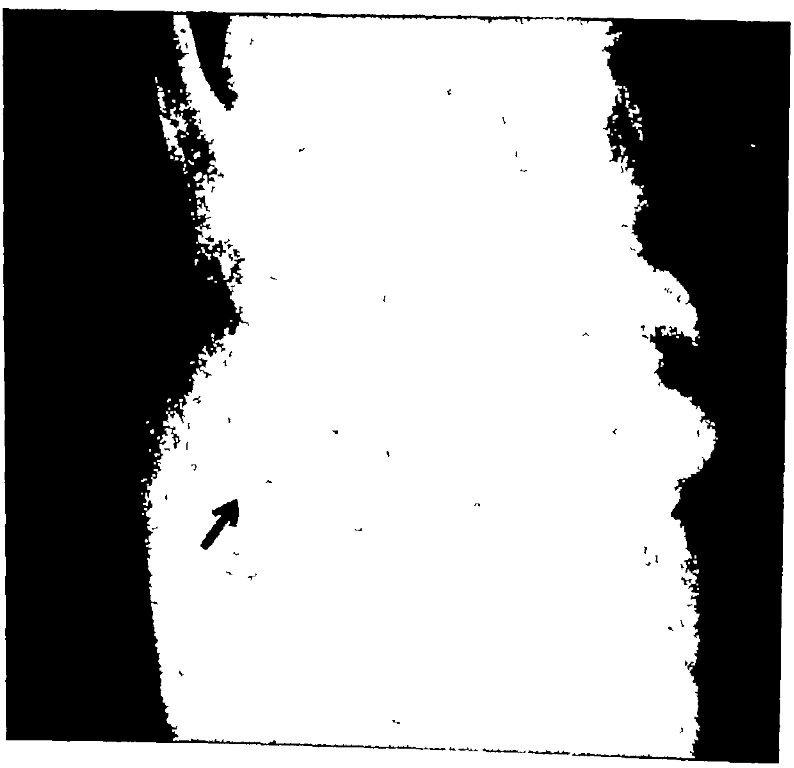
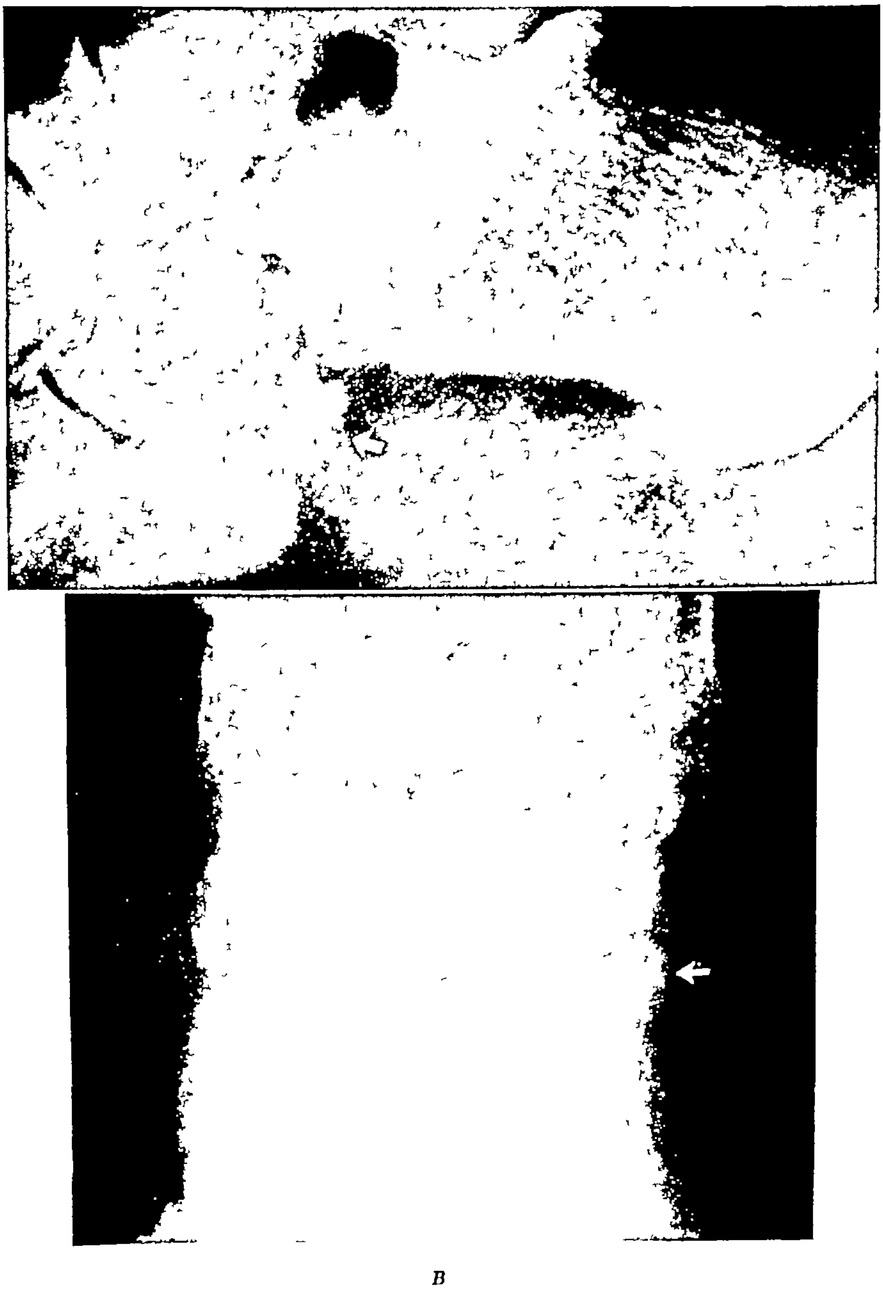


Fig 287 —Avulsion fracture of neck of navicular and fracture of head of calcaneus

The essentials in the treatment of this type of fracture are prompt reduction of the displacement by manipulation or open reduction and internal fixation to be maintained by a well-padded plaster cast Reduction and fixation must be particularized for each case The leg should be elevated for a week or two and weight bearing not attempted for six to eight weeks

Fractures of the navicular bone are of three types (1) fracture of the tuberosity, (2) chip fracture of the dorsum, (3) compression fracture and dis-

A

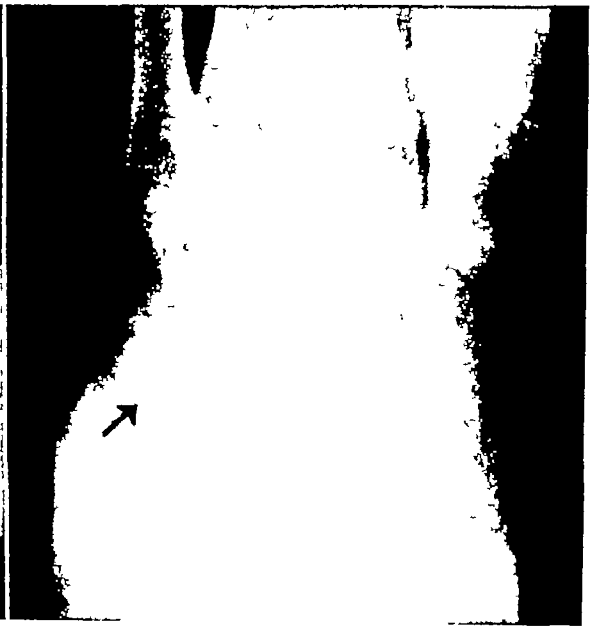


B

Fig 285 —*A*, Fracture and displacement of plantar surface of cuboid *B*, Chip fracture, lateral side of cuboid

location of the entire bone Fracture of the tuberosity of the navicular (Fig 287) must be distinguished from an accessory navicular, also known as *os naviculare, prehallux* (Kidner, 1933), and *os tibiale externum* In a true accessory bone the separation is smooth, although occasionally the separation may resemble a fracture However, the history of sudden injury with intense pain and swelling over the tuberosity, in addition to evidence of a fracture line in the roentgenogram, may be taken as evidence of a fresh fracture.

A



B

C

Fig 289 — *A*, Chip fracture on dorsum of head of talus *B*, Chip fracture, lateral side of head of calcaneus *C*, Chip fracture, anterior lip of distal end of tibia

Fig 288 —*A*, Fracture of neck of navicular, without displacement *B*, One year later, fractured fragment tilted on its end, producing a peroneal spastic flatfoot Note rarefaction at head of talus *C*, After removal of fractured fragment, peroneal spasm disappeared

or *fatigue* fracture, it is always due to the same forces unrelenting strain, pull, pressure, or stress, which finally breaks the bone (similar to metal fatigue) on which those forces have been exerted When the forces are exerted on metatarsals, especially the second, third, or fourth, the fracture is ordinarily referred to as *march fracture,* when on the first or fifth, it may be called *stress* or *spontaneous fracture,* when on the outer malleolus, usually *fatigue fracture,* sometimes *skater's fracture*

A spontaneous fracture may also occur at the base of the fifth metatarsal from severe pull or spasm of the peroneus brevis muscle which is inserted into it This is spoken of as a *stress* fracture

A B

Fig 290 —*A,* March fracture at neck of second metatarsal *B,* March fracture, comminuted with extensive formation of callus and coincidental aseptic necrosis of fourth digit

Symptoms.—The onset of symptoms may be so insidious that relief is not sought for weeks later The patient may notice swelling, tenderness, and enlargement over the fracture, partly caused by callus around the fracture Weight bearing is painful

Diagnosis —Roentgenograms generally reveal the fracture, however, the break may be only an infraction, the fracture line may be so fine that the roentgenologist's report may be negative, especially regarding early cases In late cases, proliferative callus around the shaft always indicates the pathologic condition

Such fractures have been mistaken for sarcoma Dodd (1933) reported one case of amputation of the foot in which *march* fracture was mistaken for sarcoma

Immobilization in a plaster cast, with the foot in mild plantar flexion and inversion for six weeks, will bring about healing in most instances In an exceptionally large navicular or a bifurcated navicular, the tendon of the tibialis posticus may be attached mainly to the tuberosity, and, as a result, the pull of this muscle may rotate the fractured fragment on its end (Zadek, 1926), causing persistent irritation at the medial side of the head of the talus and a compensatory peroneal spasm That is what happened in the case of a woman, aged 53 years, who had incurred a fracture of the tuberosity of the navicular ten months previously (Fig 288, *A*), for which her foot had been immobilized in a walking cast for six weeks After removal of the cast, pain gradually returned and increased in intensity, accompanied by a gradual development of a peroneal spastic flatfoot Two consultants had advised a triple arthrodesis On examination, the fractured tuberosity was observed to be tilted on its end (Fig 288, *B*), causing irritation of the head of the talus The irritation caused the compensatory peroneal spasm After removal of the fragment (Fig 288, *C*), the foot returned to normal Excision of the detached tuberosity ordinarily returns the foot to normal

Chip fractures over the dorsum of the navicular are not uncommon, they are common on other bones of the foot (Fig 289) In most instances, strapping of the foot is sufficient to heal the part At times the foot must be immobilized in a cast for a few weeks

The *body of the navicular* fractures in either a transverse or a vertical plane Displacement of the fragments is the rule one dorsal and one plantar or one large fragment displaced medially The fracture often shows some compression In most instances, it is possible to force the fragments into position by pressure over the dorsum of the navicular while the foot is held in plantar flexion, however, it is often difficult to maintain the fragments in reduction even after a molded cast has been applied (Day, 1947) Such cases require countertraction applied by inserting a pin through the calcaneum and a Kirschner wire through the base of the metatarsals The pin and wire are incorporated in the plaster cast In severe cases arthrodesis of the talonavicular and the navicular first cuneiform joints is indicated

SPONTANEOUS FRACTURES

Fatigue fracture *(march fracture* or *stress fracture)* is a spontaneous fracture of normal bone which takes place when several forces, harmless when acting independently, combine to cause the fracture The foot, more than any other part of the skeleton, is subject to such a fracture

The second metatarsal is ordinarily the longest and is the bone that assumes most of the burden when the first metatarsal is inefficient This may explain why the second metatarsal is the one likely to fracture spontaneously Osteoporosis, such as is seen in Sudeck's atrophy (page 305), also predisposes to spontaneous fracture

Terminology.—*March* fracture is ordinarily thought of as occurring in adults, especially soldiers, however, *fatigue* fracture and *march* fracture are essentially the same Regardless of whether the spontaneous fracture is called *march, stress,*

Sarcoma of the metatarsal bone is rare, march fracture is frequent. One should be wary, therefore, of making a diagnosis of malignancy of the metatarsal bones

March Fracture—March fracture of the metatarsal bones is sometimes referred to as *Deutschlander's disease* or *pied forcé* (Fig 290) Such a fracture of the proximal third of the fifth metatarsal is called *Jones' fracture* (Watson-Jones, 1952) It is common, it involves the second metatarsal bone most and the third next in frequency, but it may affect any of the lesser metatarsals, although the first only rarely The fracture line is at the neck or middle third of the shaft but may occur at any part of the metatarsal

Breithaupt (1855) was first to describe the occurrence of a "persistent swelling and pain in the forefoot," which he had observed among German soldiers after prolonged marches Thereafter, however, the malady was thought to be due to general disease or to be of neoplastic origin Stechow (1897) first studied the condition roentgenologically and recognized it as a fracture During World War I, the disorder became familiar. Maseritz (1936) studied the published reports exhaustively Although the fracture is encountered among civilians, it is essentially a disorder recognized among soldiers. The cause is not clear except that it is observed after long marches or after an extraordinary episode of prolonged standing or strenuous exercise On the other hand, all five of Singer and Maudsley's (1954) cases were of middle-aged and elderly patients who had not undergone unusual activity This would lend support to the theory advanced by Maseritz that calcium disturbances in the bone, a likely condition in the aged, might be etiologic His case presented fragmentation of the internal cuneiform, fractured head of the second metatarsal bone, and fractured base of the fifth metatarsal—all pathologic changes not ordinarily ascribed to march foot

Watson-Jones suggested that when the head of a short first metatarsal bone is in line with the neck of the second and third metatarsals, too much weight is thrown upon the heads of the second and third metatarsal bones, thereby predisposing the second to march fracture at its neck The fracture, however, occurs as frequently on the shaft of the metatarsal as it does at the neck

Undoubtedly normal variations in pattern and relationship of the metatarsal bones to one another have a bearing on excessive stress exerted on one metatarsal over the others Deformities, such as claw toes or hallux valgus, can also shift undue weight thrust to one of the metatarsal bones

Spontaneous Fracture of Metatarsals in Children—Spontaneous fracture of the metatarsals in children is uncommon but not really rare, notwithstanding the general impression that it is limited to adults Childress (1946) reported a case of his own His review of published reports disclosed only one other case, reported by Zeitlin and Odessky (1935) Popp (1953) reported six cases Three personal cases were in children (Fig 291)

In cases without displacement, which is usual, partial immobilization with adhesive or elastic anterior metatarsal arch band in addition to a shoe with a semirigid sole will suffice In the rare case in which displacement takes place, it is necessary to resort to reduction and complete immobilization

Fatigue or Stress Fracture of Malleoli—*Spontaneous or stress fracture of the lower tip of the fibula,* sometimes called *skater's fracture,* may occur at any age,

A

B

C

Fig 291 —*A*, March fracture of third metatarsal of child *B*, Spontaneous fracture of neck of fifth metatarsal of infant *C*, Spontaneous fracture of fourth metatarsal of child (*C*, Courtesy Dr Frank Weinstein)

of a laced Oxford for six to eight weeks will usually suffice Sometimes a walking cast is necessary to alleviate pain.

AVULSION FRACTURE OF CALCANEAL TUBEROSITY

Avulsion of the calcaneal tuberosity is rare in children, but rarer in adults When it occurs in children, it is before fusion of the epiphysis has taken place (Struppler, 1937, Rothberg, 1939) Extreme contraction of the calf muscle during violent exercise causes the injury, which is characterized by severe sudden pain and inability to plantar-flex the foot

Treatment invariably requires open reduction and fixation, because there is no other safe way to maintain the fragment that is still attached to the tendo achillis For the same reason, in most instances the tendo achillis often requires lengthening before reduction is possible. Mooney (1935) was able to maintain the reduction by interrupted chromic sutures around the margins of the fracture. It is probably safer, however, to insert a pin through the fragment and body of the calcaneus The pin is incorporated in a plaster cast for eight weeks, during which weight bearing is not permitted.

Major fractures of the calcaneus and talus are always complicated problems They have been fully discussed in all standard textbooks on fractures and orthopedic surgery

DISLOCATIONS

A *dislocation* means that articular surfaces of the bones forming a joint have been displaced from their normally articulating position Tearing and stretching of the periarticular structures, which are the ligaments, joint capsule, tendons, nerves, and vessels coursing over the joint, are associated with the displacement process Dislocation in the foot is often accompanied by chip fracture Dislocations may be acute, recurrent, or old unreduced or static

Sudden dislocation of the major joints of the foot without fracture seldom happens (Fig 293), static or old dislocations, such as of the hallux valgus and second metatarsophalangeal joint, are common Symptoms of *acute* dislocations are swelling, severe pain, deformity, false motion, or new motion (Symptoms and treatment of static or old unreduced dislocations are discussed in Chapter 16, Static Deformities)

Treatment of *recurrent dislocations* of the ankle is suggested in Chapter 5, under Sprains Most acute dislocations of the foot can be reduced by closed manipulation under anesthesia Only occasionally do they require open reduction Open reduction should not be undertaken until conservative treatment has been exhausted, unless the injury is complicated by interposition of tissues between the articular surfaces, which prevents manual reduction, or unless there is injury to a blood vessel or nerve of the region

Sudden interphalangeal or metatarsophalangeal dislocations are uncommon (Fig 294) They can be readily reduced, occasionally local anesthesia is required, and, for children, sometimes a general anesthetic is necessary. Dislocation of the first metatarsophalangeal joint usually resists closed reduction.

especially, however, among young athletes and in middle-aged women Griffiths (1952) records eight cases among children The condition is frequently misdiagnosed or not discovered for weeks after the onset. Bonnin (1950) records a case of an athlete who dropped to the ground at the finishing post because of painful ankle, the fracture was not discovered until four weeks after the original injury Burrows (1940) reported a case and suggested that this type of fracture is not rare He urged further reports, which were forthcoming, so that by 1948 he was able to record ninety published and personal cases Since then, many more reports have been published, notably the fifty cases among athletes recorded by Devas and Sweetnam (1956)

The fracture, as a rule, takes place about 2 to 6 cm above the tip of the fibular malleolus (Fig 292), however, it may occur at any level of the fibula. The fracture at the lower end of the fibula is usually transverse and may be complete, with or without displacement, or it may be incomplete *(infraction)* An infraction may be overlooked in the early stage because the fracture line may be faint, becoming pronounced only after a few weeks when subperiosteal thickening has taken place

Fig 292 —Fatigue fracture of lateral malleolus

The symptoms are pain and swelling over the outer malleolus, of insidious onset without apparent reason In the early stages roentgenograms may or may not readily reveal a bone lesion After a few weeks subperiosteal bone thickening and the line of fracture become apparent

A comparable fracture of the medial malleolus is less common Singer and Maudsley (1954) reviewed and reported five such cases

In the treatment of stress fracture of the outer and medial malleolus, support of the ankle with adhesive strapping and an elastic ankle support and wearing

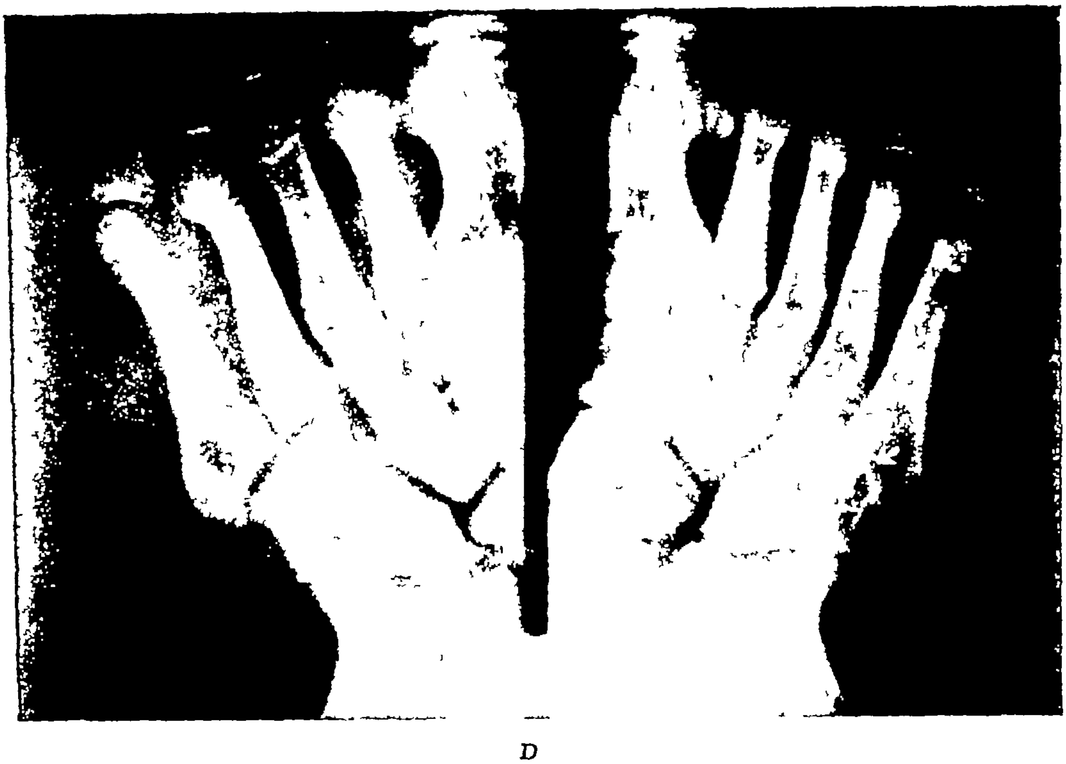
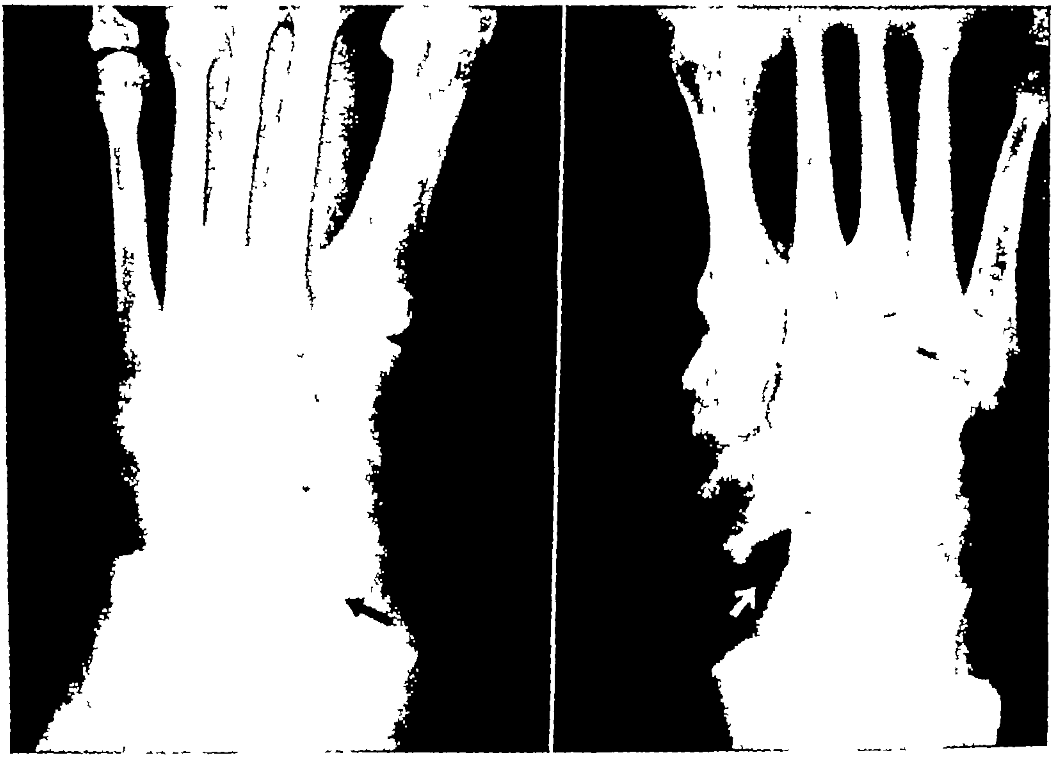


Fig 293(cont'd) —For legend see opposite page

Fig 294 —Complete sudden dislocation, without fracture, of distal part of fifth toe



A *B*

C

Fig 293—*A*, Acute dislocation of talonavicular joint *B*, Old unreduced dislocation of talonavicular joint *C*, Acute dislocation of talonavicular joint *D*, Right, Dislocation of base of fifth metatarsal cuboid joint Left, Opposite foot normal, for comparison

2 Retract the margins, including the extensor tendons, and introduce a bone elevator between the articular surfaces With traction on the foot and leverage on the elevator, force the bones into normal position

3 If the joint is unstable, wire or pin fixation may be used

4 Apply a plaster splint with the ankle at 90 degrees The splint is maintained for four weeks, after which the patient wears a walking cast for another month and for the ensuing six months, after removal of the cast, a well-fitted shoe with a semirigid arch support and sturdy soles The wire or pin may have to be removed Removal is permissible any time after removal of the plaster cast

REFERENCES

Bonnin, J G Injuries to Ankle, New York, 1950, Grune & Stratton, Inc, p 260

Breithaupt Zur Pathologie des menschlichen Fusses, Med -Ztg 24 169, 175, 1855. (Cited by Maseritz)

Burrows, H J Spontaneous Fracture of Apparently Normal Fibular at Its Lower Third, Brit J Surg 28 82, 1940

Burrows, H J Fatigue Fracture of Fibula, J Bone & Joint Surg 30B 266-279, May, 1948

Campbell, see Speed and Knight

Childress, H M March Foot in a Seven-Year-Old Child, J Bone & Joint Surg 28 877, Oct, 1946

Day, A J Treatment of Injuries to Tarsal Navicular, J Bone & Joint Surg 29 359, April, 1947

Devas, M B, and Sweetnam, R Stress Fractures of the Fibula, Review of Fifty Cases in Athletes, J Bone & Joint Surg 38B 818, Nov, 1956

Dodd, H Pied Forcé or March Foot, Brit J Surg 21 131, July, 1933

Easton, E R Two Rare Dislocations of Metatarsals at Lisfranc's Joint, J Bone & Joint Surg 20 1053, Oct, 1938

Gissane, William A Dangerous Type of Fracture of the Foot, J Bone & Joint Surg 33B 535-538, Nov, 1951

Griffiths, A L Fatigue Fracture of Fibula in Childhood, Arch Dis Childhood 27.552-557, Dec, 1952

Hermel, M B, and Gershon-Cohen, J Nutcracker Fracture of Cuboid by Indirect Violence, Radiology 60 850-854, June, 1953

Holstein, Arthur, and Joldersma, R D Dislocation of First Cuneiform in Tarsometatarsal Fracture-Dislocation, J Bone & Joint Surg 32A 419-421, April, 1950

Kidner, F C Prehallux in Relation to Flatfoot, J A M A 101 1539, Nov 11, 1933

Manuel del Sel, Jose Surgical Treatment of Tarso-Metatarsal Fracture-Dislocations, J Bone & Joint Surg 37B 203, May, 1955

Maseritz, I H March Foot Associated With Undescribed Changes of Internal Cuneiform and Metatarsal Bones, Arch Surg 32 49-64, Jan, 1936

Mooney, Voight Avulsion of the Epiphysis of the Os Calcis, J Bone & Joint Surg 18 1056-1057, Oct, 1935

Popp, O Schleichende Metatarsalfrakturen bei Kindern, Wchnschr Orthop 84 55, 1953 (Abstracted, Surg Gynec & Obst 98 5, May, 1954)

Rothberg, A S Avulsion Fracture of the Os Calcis, J Bone & Joint Surg 21 218-220, Jan, 1939

Singer, Martin, and Maudsley, R H Fatigue Fracture of Lower Tibia, Report of Five Cases, J Bone & Joint Surg 36B 647-651, Nov, 1954

Speed, J S, and Knight, R A Campbell's Operative Orthopaedics, ed 3, St Louis, 1956, The C V Mosby Co, p 508

Stechow Fussodem und Roentgenstrahlen, Deutsche mil -arztel Ztschr 26 465, 1897 (Cited by Maseritz)

Stechow Fussodem und Roentgenstrahlen, Internat Cong f Hyg u Demogr 8 99, 1898 (Cited by Maseritz)

Struppler, Viktor Abrissbruch am Fersenbein, Bruns Beitr klin Chir 166 402, 1937

Watson-Jones, Reginald Fractures and Joint Injuries, ed 4, Baltimore, 1952, Williams & Wilkins Co, p 345

Zadek, I Significance of Accessory Tarsal Scaphoid, J Bone & Joint Surg 8 618, July, 1926

Zeitlin, A A, and Odessky, I N Pied Forcé or Deutschlander's Disease, Radiology 25 215, 1935

Open Reduction of Dislocation of First Metatarsophalangeal Joint.—The base of the proximal phalanx is usually displaced on the dorsum of the first metatarsal head The head of the metatarsal may penetrate the plantar capsule and become caught in the capsule and flexor tendons which prevents closed reduction

1 Make a semilunar incision about 8 cm long over the mediodorsal aspect of the first metatarsophalangeal joint with its vertex dorsally

2 Free and retract the skin margins Free the medial skin margin completely from the joint, including the plantar surface Often the structures that resisted closed reduction reveal themselves, so that they can be retracted and the dislocation can be reduced

3 If that is not possible, incise the capsule longitudinally at its mediodorsal aspect and retract the margins

4 Guide the base of the proximal phalanx into the joint by means of a small elevator while applying traction to the hallux

5 Completely immobilize the foot for three weeks After that period the patient should wear a shoe with a semirigid sole for about two months

Open Reduction of Dislocation of Tarsometatarsal Joints.—Any one of the metatarsals or all of them may be dislocated at the base The middle three are usually dislocated dorsally, because of their keystone-shaped base, but they may be dislocated plantarwise The first may also be dislocated medially and the fifth, laterally In most instances, such dislocations can be reduced by traction and manipulation, usually under general anesthesia, and maintained by immobilizataion in plaster When closed reduction fails, however, open reduction is inevitable

1 Make a longitudinal incision about 8 cm long over the dorsum of the dislocated joint, down through the capsule and ligaments

2 Retract the margins With a small elevator placed in the articulation, force the bones into the joints by traction and leverage

3 If the reduced joint is unstable, insert a wire, pin, staple or screw for fixation

4 Apply a plaster splint from the toes to the knee with the foot in mild plantar flexion

5 In three weeks, apply a walking cast that is well molded in the longitudinal arch The patient wears the walking cast for four weeks and after that a rigid-soled shoe with a longitudinal inlay for about three months

Open Reduction of Dislocation of Midtarsal Joints—Midtarsal dislocation without fracture is uncommon Of those that do occur, the navicular is most commonly displaced and is almost always displaced either medialward or dorsally

Most acute dislocations of the midtarsal are difficult to reduce by closed reduction and difficult to maintain in reduction even when reduction is possible This applies especially to the navicular Closed reduction, nevertheless, should be attempted first and open reduction and fixation resorted to only when it fails The following technique is recommended

1 Make an 8 cm longitudinal incision over the dislocated joint, carrying it down through the ligaments and capsule

tarsus is made up of short heavy bones, and because the normal movement in the tarsal joints is limited inasmuch as the articular surfaces of the tarsal joints are comparatively flat

On the other hand, the phalanges and metatarsals are long thin bones having normally wide range of joint motion Their type of restrictive force produces most of the static deformities of the forefoot hallux valgus, hammertoe, tailor's bunion, overlapping toes, clawtoes, and many other deviations from normal

The type of restriction exerted by a shoe determines the deforming effect It should be self-evident that a narrow or tight shoe, which produces a transverse restriction on the foot, cannot cause static deformities as is sometimes asserted Any severe transverse constriction over the foot sufficient to produce deformity would act as a tourniquet, the acute pain would be unbearable, the prolonged and repeated constriction would result in acute vascular impairment rather than in skeletal deformity

A well-developed strong foot withstands surprising abuse, morbid changes take place only when maltreatment becomes excessive An underdeveloped and frail foot may fail under ordinary stress and strain

MORTON'S SYNDROME

A short first metatarsal, a hypermobile first metatarsal segment, and a posterior displacement of the sesamoids comprise the syndrome described by Morton in 1935 which carries his name. The syndrome results in hypertrophy of the second metatarsal, tenderness at the base of the second metatarsal, and callosities under the heads of the second and third metatarsals Morton observed that a high percentage of weak feet and metatarsalgia is directly related to the syndrome He thought that the hypermobility of the first metatarsal bones is due to its shortness, which permits abnormally free motion in the joint between the first cuneiform and navicular bones and between the first and middle cuneiforms The resulting instability is reflected in malfunction of the metatarsal and of the longitudinal arch of the foot

Harris and Beath took issue in 1949 with Morton's interpretation, nevertheless, in 1952 Morton again supported and enlarged his theory Statistical studies by Hawkes (1914), among others, regarding the relative length of the metatarsals demonstrated that in about 80 per cent of human feet the first metatarsal is shorter than the second In agreement with Jones (1944), "The only alternative is to assume that there is such a thing as ideal foot function and that this function could presumably be carried out by an ideal but not by the normal foot"*

HAMMERTOES (CLAWTOES)

Hammertoe or clawtoe, which is essentially a partial or complete dislocation of the interphalangeal joints, is common and may occur in more than one toe The dislocation is usually a dorsal one, but occasionally it may be plantarwise In the lesser toes, the proximal joint is frequently involved, the distal joint only

*From Jones, F W Structure and Function as Seen in the Foot, Baltimore, 1944, Williams & Wilkins Co

CHAPTER SIXTEEN

Static Deformities

ANY PART OF THE SKELETAL SYSTEM MAY BECOME DEFORMED AS A RESULT OF A sudden or rapid alteration when trauma or disease disrupts normal function of a skeletal unit. Unrelenting stress or pressure on a given part is a second cause of static deformity. Whenever distortion or any underlying cause of malformation remains untreated, the deformity becomes fixed or static, the anatomic structures affected slowly accommodate themselves and become altered according to Wolff's Law (1884):

> *Every change in the use or static function of bone causes a change in its internal form and architecture as well as alterations in its external formation and function, according to mathematical laws.*

The foot more than any other skeletal unit is subject to static deformities. Its weight-transmitting and propulsive functions are restricted daily by nonyielding, unscientific foot covering; consequently, the normal tissue changes its shape and ability, according to Wolff's Law and Davis' Law. (See page 53.) It is well recognized that restrictive or expansive forces during the age of growth can alter any part of the body. We know what happens to African natives who elongate their lips and ears; we recall the spinal deformities caused by the constriction of Victorian corsets and the deforming of the female foot by the Chinese practice of binding it (Fig. 47). Straightening of congenitally irregular teeth by the orthodontist is an example of healthful alteration by applied force in the right direction. In our society the commonest restrictive force is short shoes; the heel of the shoe acts as an anchor point and most of the containment of the forepart of the shoe is accomplished by exerting force against the distal end of the toes. The toes in turn exert some of that pressure against the metatarsals, and, to some degree, the metatarsals exert some of the restriction on the tarsus. However, the restrictive force produces little static deformity on the tarsus, because the

distal phalanx (Fig 296) The dorsal capsule is stretched and the plantar capsule is contracted The extensor hallucis longus is also contracted

Three Middle Toes—The head of the proximal phalanx is displaced dorsally The base of the middle phalanx articulates with the plantar surface of the head of the proximal phalanx (Fig 297) The dorsal capsule is stretched and the plantar capsule is contracted The tendons of flexor digitorum longus and extensor digitorum longus are contracted Comparable displacement is present when the dislocation is in the distal phalangeal joint, however, there the deformity is between the middle and distal phalanx

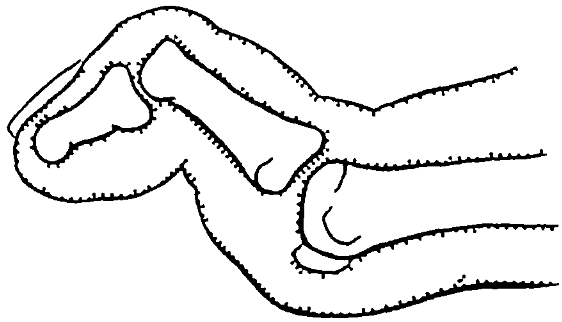


Fig 296—Hammered great toe Note articulation of distal phalanx with plantar surface of head of proximal phalanx

Fig 297—Hammered second toe Note articulation of middle phalanx with plantar surface of head of proximal phalanx

Fifth Toe—The entire toe becomes semicircular The middle phalanx is a keystone in an arch formed by the three phalanges The plantar capsules are contracted The dorsal capsules are stretched

Treatment.—Many complex procedures have been offered for the correction of hammertoes, especially of the middle three toes (Fig 298) Taylor (1940) and Selig (1941) suggested drawing a Kirschner wire through all the phalanges

rarely In the fifth toe, it is usually both the proximal and distal joints that are affected, so that the toe becomes semicircular. Hyperextension of the metatarsophalangeal joint is the result of overaction of the extensor muscles

Etiology.—Hammertoe deformity of the great toe is often due to severing of the tendons of the flexor hallucis longus, the flexor hallucis brevis, or both, on excision of the sesamoids, sometimes performed for reduction of hallux valgus In such cases, disabling is serious The deformity is caused by the powerful pull on the extensor hallucis longus The great toe assumes as much burden in walking as the four other toes do together, furthermore, the plantar surface of the great toe joint is a weight-bearing pivot

Congenital—Congenital hammertoes are usually multiple and are associated with the different types of talipes equinus or clawfoot Occasionally a single toe may be congenitally hammered When it is, it is bilateral and ordinarily is in the second toe (Fig 295)

Fig 295 —Congenital bilateral hammered second toe

Acquired—Long flaccid toes and prolonged wearing of short footgear are the common causes of acquired hammered toe in the second, fourth, and third toes of one or both feet, in that order of frequency of location The second toe is normally the longest and therefore receives most of the containment pressure of short shoes, the fourth toe receives most of the pressure of pointed or inflare shoes or both

Structural Changes.—The great toe, the three middle toes, and the fifth toe undergo a pattern of structural alterations

Great Toe—The head of the proximal phalanx is displaced dorsally, and its inferior surface lies immediately above the articular surface of the base of the

(Fig. 299) Tierny (1938) and Young (1938) advised forming a cup and ball of the distal end of the proximal joint Borg (1950), Michele and Krueger (1948), Lapidus (1939), and Lorenz (1929)—each excised different parts of the phalanges Wagner (1934) and Forrester-Brown (1938) advocated tendon transplantation to correct hammered great toe

Recommended Atraumatic Procedures for Reduction of Hammered Great Toe—The procedure outlined herewith is intended for mild cases, in which the deformity is not fixed and can be reduced passively, although it cannot be held in reduction

1 Section the tendon and capsule on the plantar surface of the phalangeal joint through a medioplantar incision This permits immediate reduction of the deformity

Fig 299—Hammered second toe Arthrodesis of both interphalangeal joints, pin inserted elsewhere by another surgeon One year postoperatively, digit was entirely rigid and in dorsiflexed position, requiring further surgical intervention

2 To maintain reduction, make a longitudinal incision over the dorsum of the phalangeal joint, remove a transverse spindle-shaped section, about 0 5 cm in width, from the dorsal-phalangeal joint capsule and tendon of the extensor hallucis longus

3 Suture the dorsal capsule and tendon of the extensor hallucis longus in the shortened position and suture the skin

4 Partly immobilize the toe by adhesive splinting for two to three weeks The splinting may be achieved by bandaging the great toe to the three adjacent toes and carrying the bandage over all the metatarsophalangeal joints.

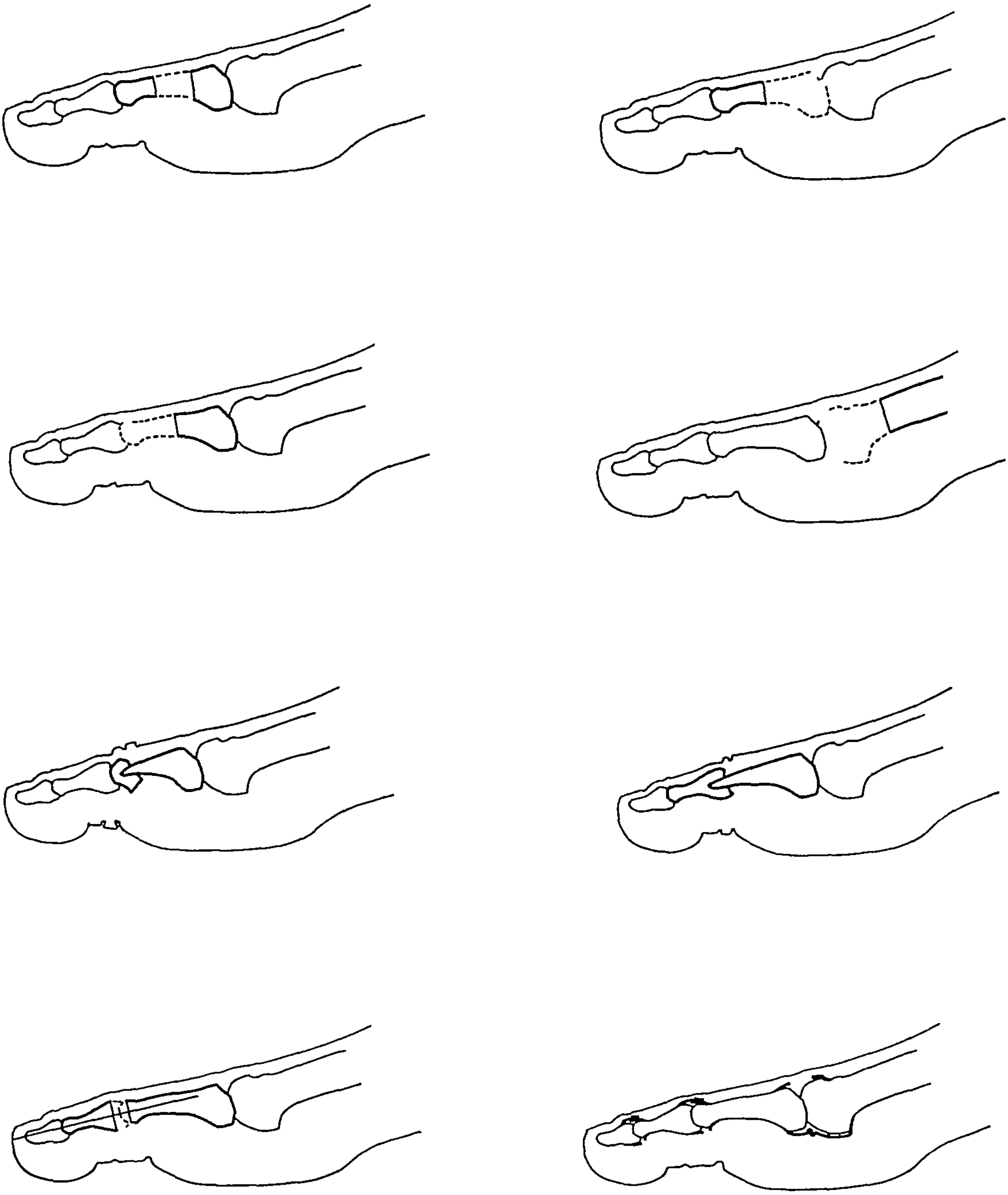


Fig 298 —Various operations devised for correction of hammered middle three toes *A*, Excision of middle third of proximal phalanx *B*, Excision of proximal half of proximal phalanx *C*, Excision of distal half of proximal phalanx *D*, Excision of head of metatarsal *E*, Shaft of proximal phalanx sharply pointed and fixed into its own head *F*, Distal end of proximal phalanx cupped into base of middle phalanx *G*, Proximal phalangeal joint denuded of cartilage Kirschner wire passed through three phalanges *H*, All contracted capsules sectioned Elongated capsules shortened

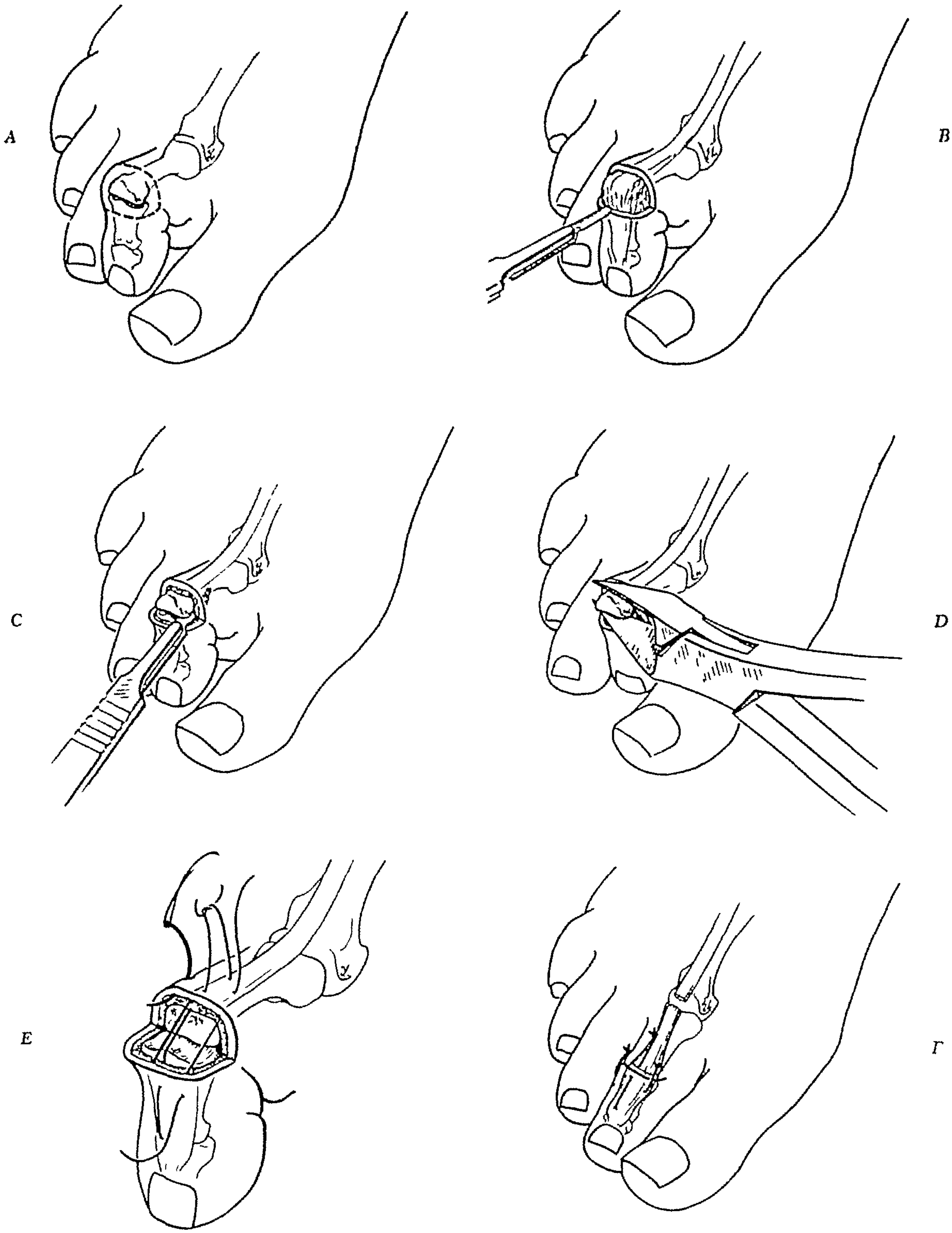


Fig 301 —Recommended procedure for reduction of hammered middle three toes *A*, Elliptical incision over the dorsum of head of proximal phalanx *B*, Like sections of tendon and capsule removed *C*, Capsule incised on both sides of head of proximal phalanx *D*, Head of proximal phalanx amputated *E* and *F*, Skin, tendon, and capsule sutured

For fixed, rigid cases, Jones' (1917) technique for transference of the extensor hallucis longus and arthrodesis of the interphalangeal joint still offers best results

1 Make a transverse incision over the hallux phalangeal joint (Fig 300, *A*)

2 Detach the extensor longus hallucis tendon from its insertion

3 Make a second longitudinal incision along the border of the extensor hallucis longus tendon, beginning over the base of the proximal phalanx and extending proximally to the middle of the first metatarsal shaft

4 Pull out the extensor hallucis longus tendon at the proximal end of this incision

5. Drill a transverse hole through the first metatarsal just behind and at the upper part of the neck.

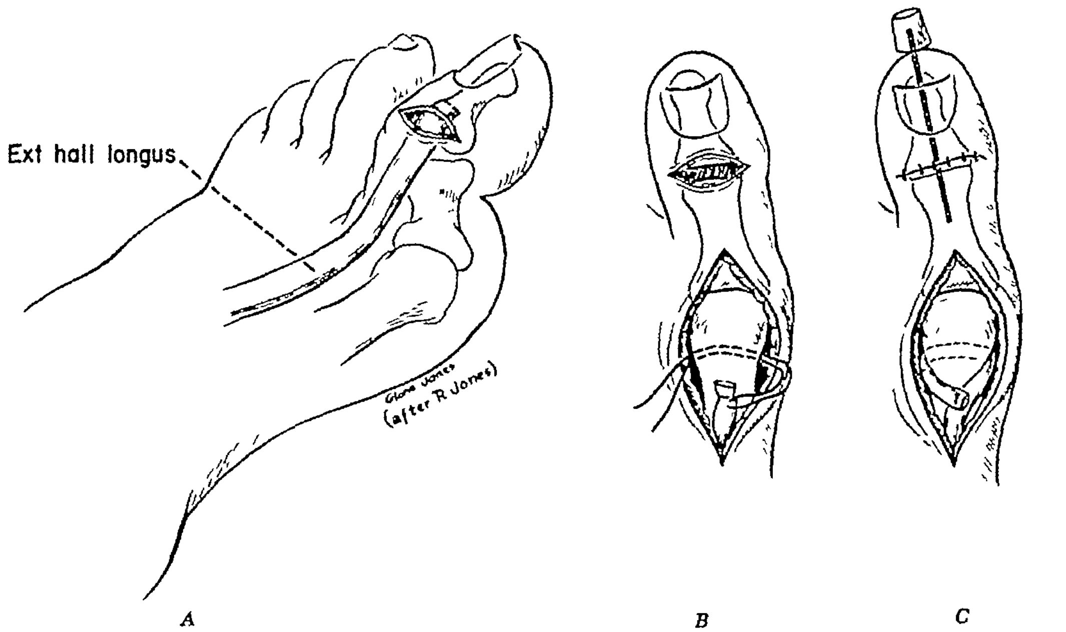


Fig 300 —Jones' reduction for static hammered great toe *A*, Clawfoot showing incision for cut and release of tendon *B*, Phalangeal joint denuded of cartilage and the tendon threaded through drill hole *C*, Tendon sutured to itself and a pin inserted into the arthrodesed phalangeal joint Exposed pin covered with cork

6 Thread stainless steel wire through the end of the previously freed tendon with a straight needle Pass the needle through the drill hole and guide the tendon through it (Fig 300, *B*)

7 Suture the end of the tendon to the body of the tendon on the dorsum of the first metatarsal shaft

8 Denude the interphalangeal joint of cartilage and flatten the articular surface so that they fit perfectly

9. Pass a Steinmann pin through the distal phalanx and half way through the shaft of the proximal phalanx, then fit a cork over the exposed part of the Steinmann pin to protect it (Fig 300, *C*)

10 Close the skin as usual and apply a plaster splint

11 In ten days remove the splint and sutures Apply a boot cast with walking heel, which the patient wears for six weeks

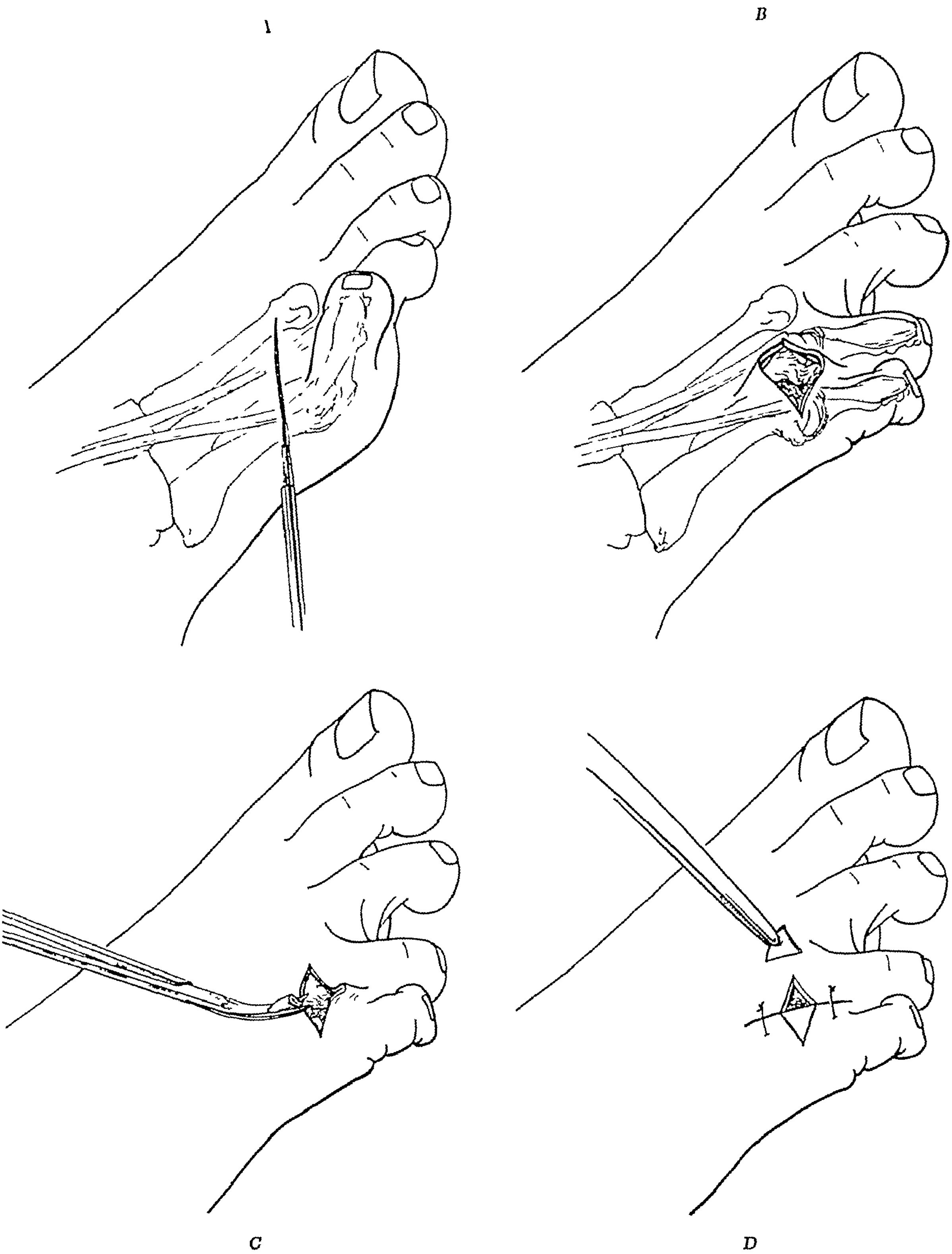


Fig 303 —DuVries' technique for reduction of hammered toe with severe skin contraction A, Transverse incision over dorsum of fourth and fifth metatarsophalangeal joints just behind the metatarsal necks *B*, Dorsal tendon and capsule of fifth metatarsophalangeal joint sectioned All other subdermal adhesive fibers severed Fourth and fifth toes plantar-flexed to form diamond-shaped incision Transverse axis narrowed Middle of both skin margins puckered *C*, Skin puckering excised, pucker forms triangular section of skin *D*, Rotation of triangular pieces of skin gives perfect fit into defect Suture in that position

Reduction of Hammered Middle Three Toes —The following procedure for reduction of hammered middle three toes is recommended because it is atraumatic

1 Make a spindle-shaped transverse incision over the joint prominence extending from one side of the joint to the opposite side

2 Remove the skin and subcutaneous tissue (Fig 301, *A*)

3 Expose the tendon and joint capsule.

4 Remove a section along the outline of the original incision (Fig 301, *B*)

5 Expose the head of the proximal phalanx, if the distal joint is involved, then expose the head of the middle phalanx

6 Free the head of the proximal phalanx (or middle phalanx in the case of the distal joint) by incising the capsule on both sides of the joint (Fig 301, *C*)

7 Amputate the head across the sulci which are present on each side of the head of the bone (Fig 301, *D*)

8 Smooth the cut surface with a rasp

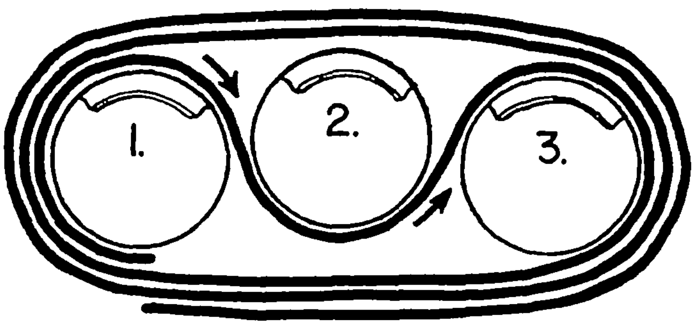


Fig 302 —Schematic drawing of immobilization of middle toe by cradle-gauze bandaging

9 Suture the margins with a mattress suture, so that the base of the stitch includes the capsule and tendon and the apex includes only the skin (Fig 301, *E-F*)

10 Occasionally, a tenotomy and capsulotomy at the metatarsophalangeal joint are necessary, so that the toe will lie flat

11 Splint the toe to the adjacent toes by a cradle type dressing (Fig 302)

Reduction of Hammered Fifth Toe —A hammered fifth toe may be corrected without trauma in the following manner

1 Make a longitudinal incision over the dorsum of the involved toe

2 Disjoint and remove a keystone section of the middle phalanx Removal of the whole middle phalanx can produce a flail toe

3 Section the dorsal tendon and metatarsophalangeal capsule when they are contracted, as they are in a high percentage of cases This step alone often corrects the disability

DuVries' Procedure for Reduction of Hammered Fifth Toe With Severe Skin Contraction A rotation skin graft is recommended

1 Make a transverse incision over the fourth and fifth metatarsophalangeal joints (Fig 303, *A*)

2 Plantar-flex the fourth and fifth digits in overcorrection This forms a proximal and distal skin puckering, the incision is diamond shaped

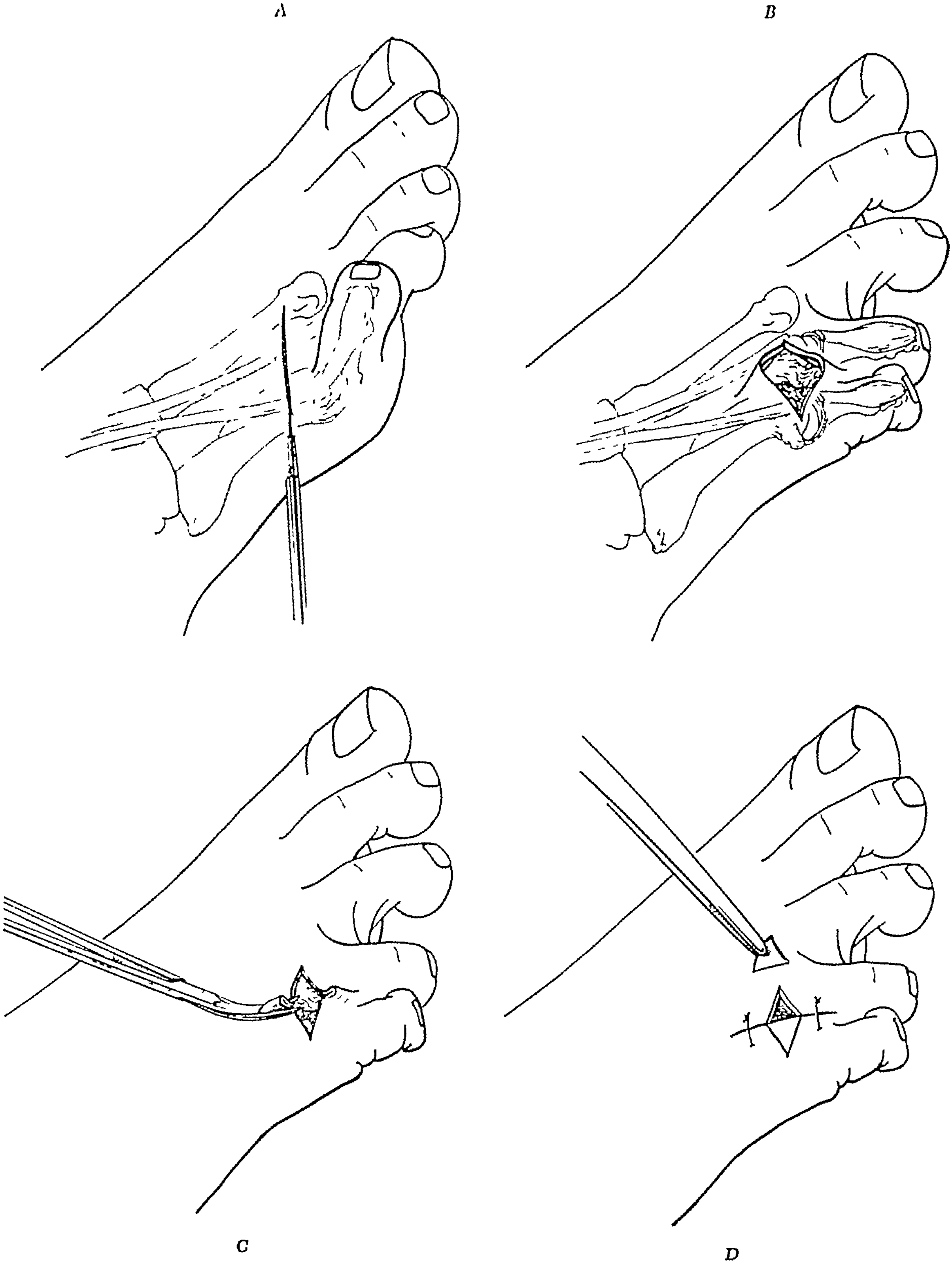


Fig 303 —DuVries' technique for reduction of hammered toe with severe skin contraction *A*, Transverse incision over dorsum of fourth and fifth metatarsophalangeal joints just behind the metatarsal necks *B*, Dorsal tendon and capsule of fifth metatarsophalangeal joint sectioned All other subdermal adhesive fibers severed Fourth and fifth toes plantar-flexed to form diamond-shaped incision Transverse axis narrowed Middle of both skin margins puckered *C*, Skin puckering excised, pucker forms triangular section of skin *D*, Rotation of triangular pieces of skin gives perfect fit into defect Suture in that position

3 Section the tendon and capsule over the fifth metatarsophalangeal joint (Fig 303, *B*)

4 Excise the triangular skin pucker (Fig. 303, *C*), rotate and fit into the diamond-shaped wound after the proximal and distal skin incisions (Fig 303, *D*) have been sutured.

5. All bleeding must be controlled because if a clot forms under the graft, the graft will not adhere.

6. The fifth digit is held in mild plantar flexion with adhesive strapping for about six weeks

OVERLAPPING TOES

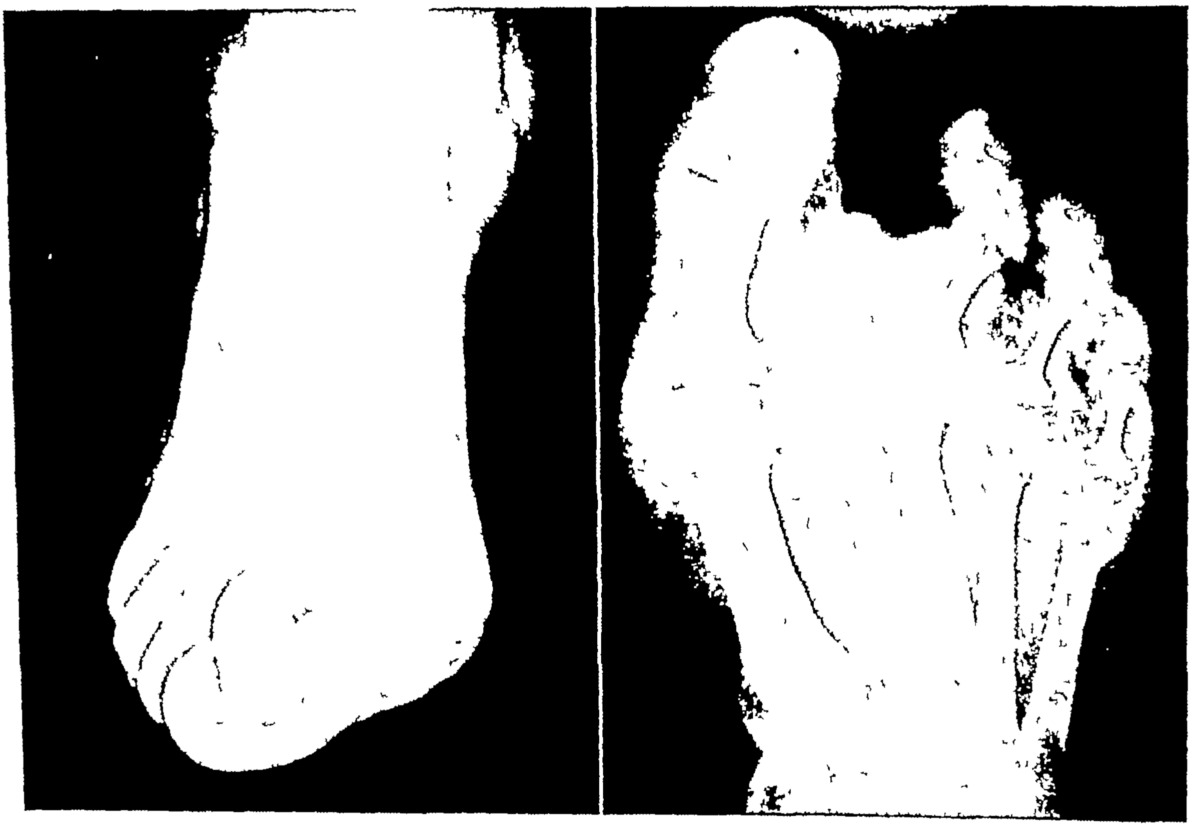
Overlapping is a condition wherein one toe lies on the dorsum of an adjacent toe, commonly the fifth toe lying over the fourth and, next in order of frequency, the second toe lying over the great toe Fifth toe overlapping (Fig 304) is usually congenital but may be acquired as a result of flaccid feet, square type of feet, the toes of which are nearly equal in length, and wearing of short shoes Contraction of the tendon of the extensor digitorum longus, of the dorsal capsule of the fifth metatarsophalangeal joint, and especially of the dorsal skin maintains the deformity

Fig 304 –Bilateral congenital overlapping of fifth toe

In second toe overlapping, in which the second toe lies over the great toe, extreme hallux valgus is usually present (Fig 305), because the second toe normally is maintained in a slight dorsiflexed attitude, whereas the great toe normally is maintained in a straight line When the great toe is forced into valgus,

it glides under the second toe The deformity is frequently coupled with dislocation of the second metatarsophalangeal joint

Lapidus' Procedure.—In 1942 Lapidus described a radical operation for the correction of fifth toe overlapping (Fig. 306) which consists of transferring the proximal end of the distal terminal portion of the extensor longus digitorum tendon of the fifth toe into the tendon of the abductor digiti quinti The procedure is performed through two incisions, one extending from the dorsal surface of the middle phalanx of the fifth toe to the lateral side of the fifth metatarsophalangeal joint The second incision is over the middle third of the fifth metatarsal. Through that incision, the extensor longus digitorum tendon is severed and the distal end of the tendon extracted through the first incision, then it is sutured to the lateral side of the fifth metatarsophalangeal joint



A B

Fig 305 —A, Overlapping second toe with hallux valgus B, Overlapping second toe without hallux valgus

Lantzounis' Procedure.—Lantzounis (1940) divides the tendon of the extensor longus digitorum of the fifth toe at a point over the shaft of the proximal phalanx and drills a tunnel behind the head of the fifth metatarsal, through which the proximal end of the severed tendon is threaded and sutured to itself

Wilson's Procedure.—Wilson (1953) advocated a procedure first described by Stamm (1948), consisting of a V-shaped incision over the base of the fifth proximal phalanx The extensor tendon and the dorsal capsule of the fifth metatarsophalangeal joint are sectioned and the fifth digit plantar-flexed This pulls

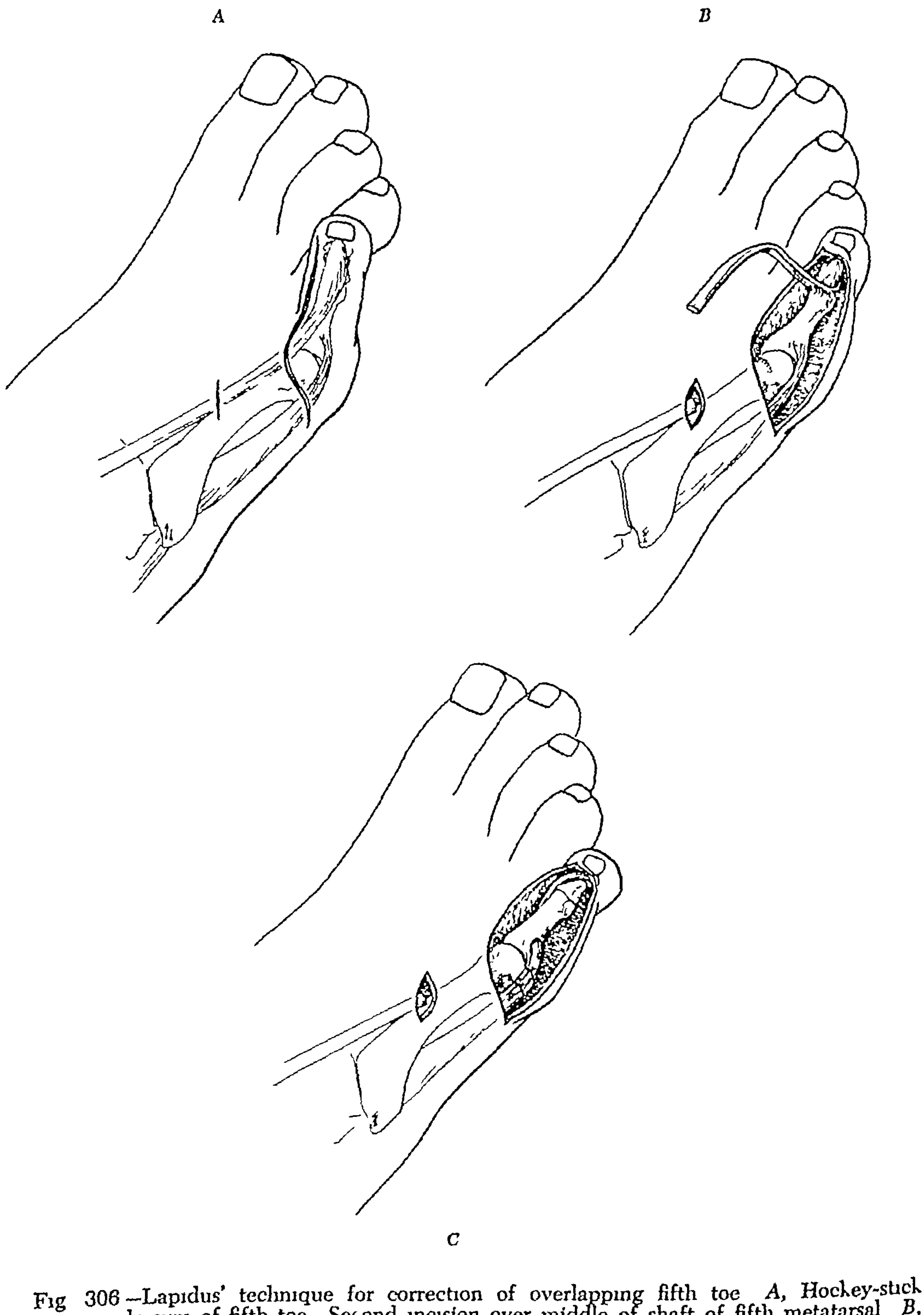


Fig 306—Lapidus' technique for correction of overlapping fifth toe *A*, Hockey-stick incision over dorsum of fifth toe Second incision over middle of shaft of fifth metatarsal *B*, Extensor digitorum longus tendon to fifth toe sectioned at second incision, distal portion retracted *C*, Freed tendon threaded through a drill hole in proximal phalanx from tibial to fibular side Sutured to abductor digiti quinti tendon

the tongue of the incision distally, now forming a Y-shaped incision The skin is sutured in the Y shape (Fig 307).

Recommended Technique for Correction of Fifth Toe Overlapping When Skin Is Not Involved.—The dorsal skin is not involved in maintaining the deformity when the fifth toe can be forcibly held in a plantar-flexed position, in such case, the following procedure is recommended·

1. Make an incision about 2 cm in length on the tibial side of the fifth metatarsophalangeal joint.
2. Section the tendon and capsule over this joint with a tenotome. At times the sectioning of the capsule needs to be carried to the sides of the joint until the toe maintains itself in a normal position.
3. Close the skin
4. Place the toe in an overcorrected position. This position is held with adhesive tape for about six weeks

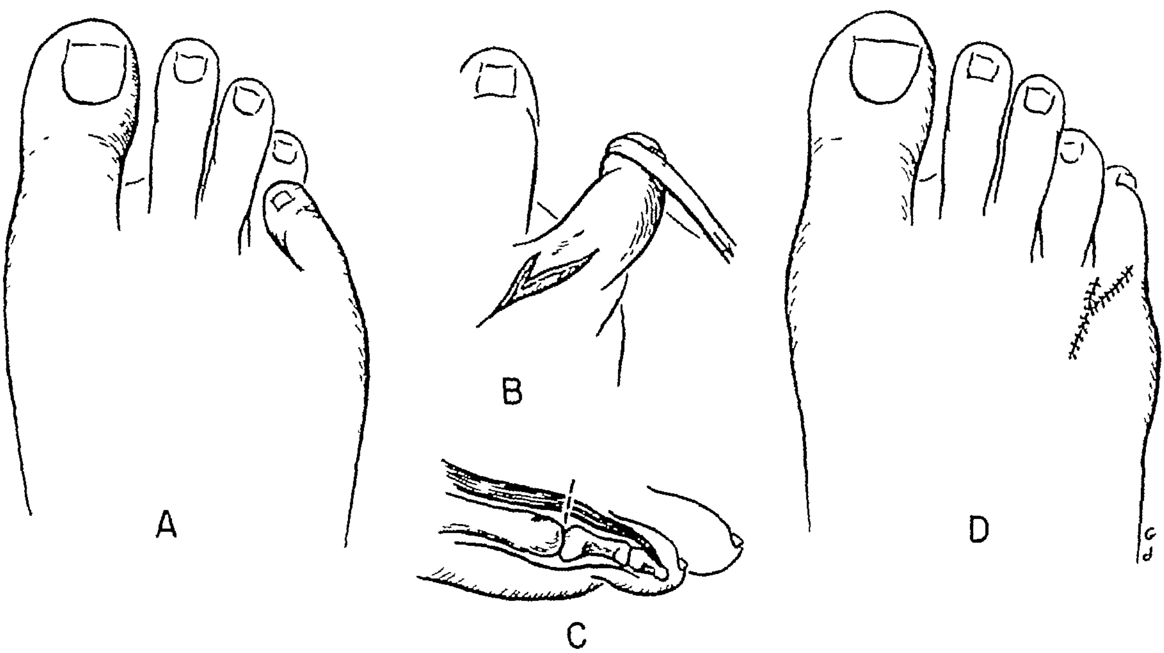


Fig 307.—Wilson's technique for overlapping fifth toe *A*, Overlapping toe *B*, A Y-shaped incision over fifth metatarsophalangeal joint *C*, Dorsal tendon and capsule of metatarsophalangeal joint, sectioned *D*, Deformity corrected, skin sutured

Recommended Technique for Correction of Fifth Toe Overlapping When Dorsal Skin Is Not Severely Contracted.—My technique for correction of overlapping of the fifth toe is recommended when the skin is not severely contracted.

1 Make a longitudinal incision over the fourth metatarsal interspace, beginning in the web between the fourth and fifth toe and extending over the dorsum to about the proximal third of the metatarsal shafts

2 Carry the incision to a depth sufficient to sever all adhesive fibers holding the toe in deformity

3 Tenotomy and capsulotomy of the dorsum of the fifth metatarsophalangeal joint are usually necessary, so that the toe can lie in a normal position by its own weight (Fig 308, *A*)

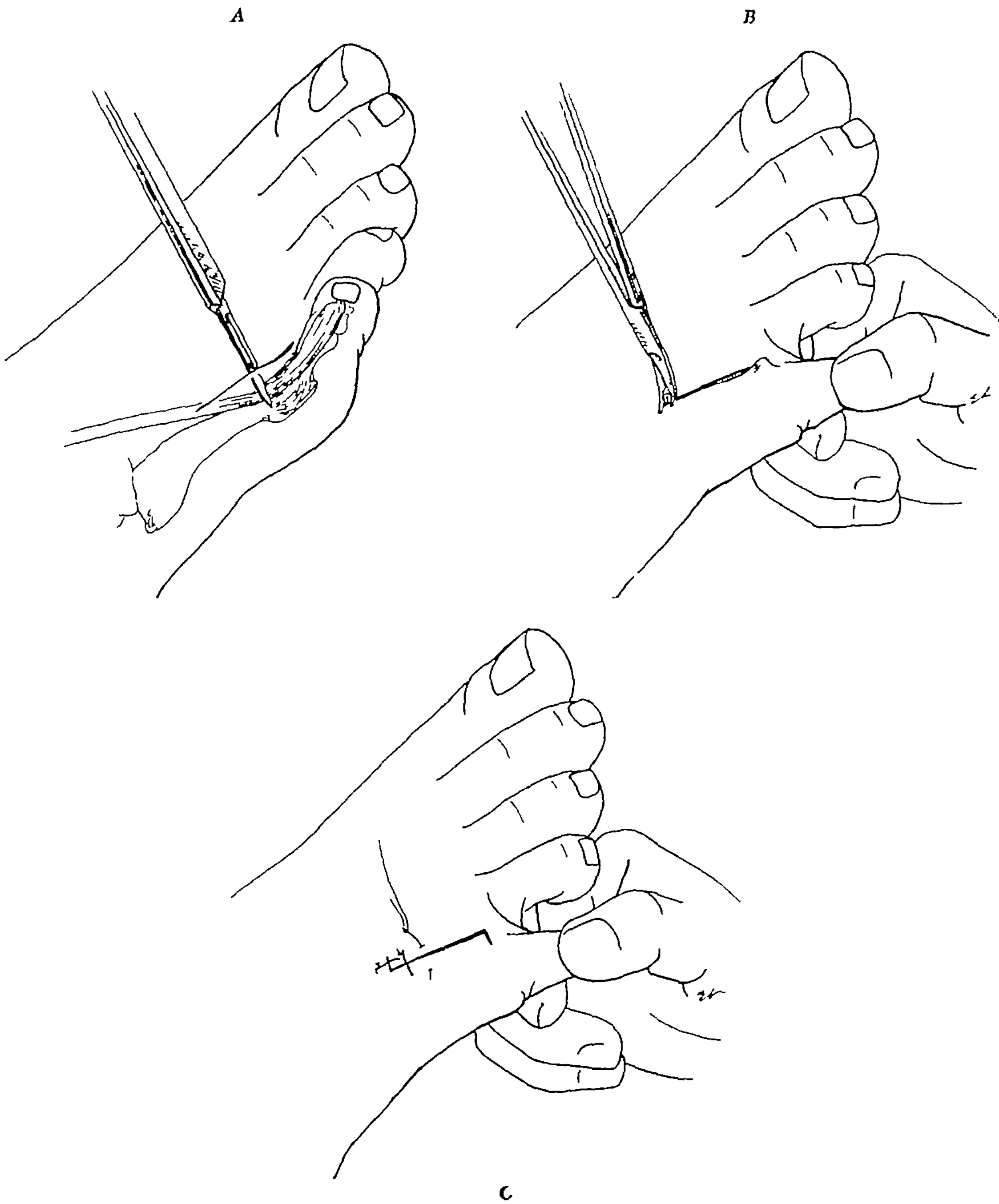


Fig 308—DuVries' technique for overlapping fifth toe without severe skin contraction (For severe skin contraction, see Fig 303, *A-C*) *A*, Longitudinal incision over fourth metatarsal interspace followed by tenotomy and capsulotomy over fifth metatarsophalangeal joint *B*, Fifth toe plantar-flexed to force fibular margin of incision distally and tibial margin proximally Forms a fold at each end of incision Folds excised *C*, Incision sutured in new position

4 By plantar flexing the fifth toe, the fibular skin margin is stretched distally and the tibial margin proximally, forming a pucker at both ends of the incision. The puckered portion is excised (Fig. 308, *B*).

5. Suture the skin margins in the new position Also suture the small transverse incision left by excision of the pucker (Fig 308, *C*).

6. The toe is held in overcorrected position with adhesive tape for about eight weeks.

Correction of Second Toe Overlapping.—The second toe always overlaps the great toe in second toe overlapping, which in most cases is static and accompanied by hallux valgus The two conditions must be reduced at the same time Overlapping may be reduced in the flaccid type of foot, when no dislocation of the metatarsophalangeal joint is present, by dorsal tenotomy and capsulotomy of the second metatarsophalangeal joint. The toe is then fixed with adhesive tape in an overcorrected plantar attitude for from six to eight weeks.

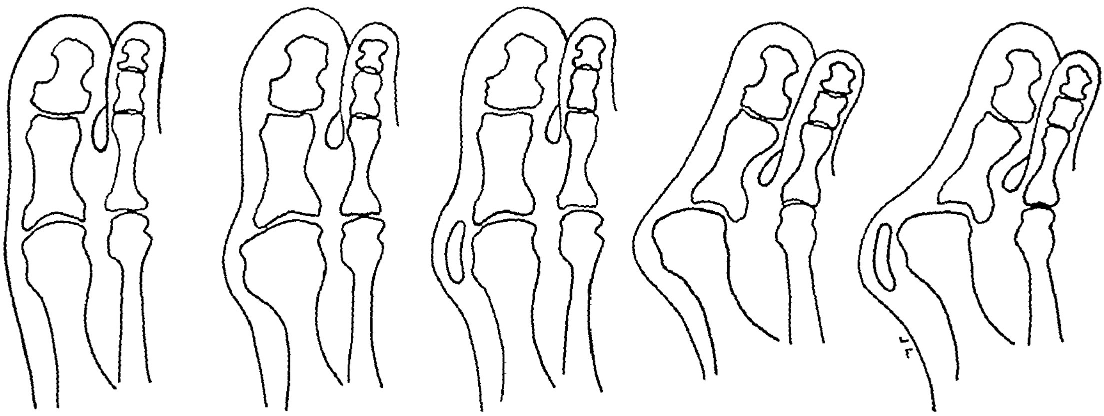
BUNIONS

The term *bunion,* derived from the Latin, *bú nio,* meaning turnip, has been confusingly misapplied to foot disorders, usually to connote any enlargement on the tibial side of the great toe joint: nonspecific chronic inflammation; chronic thickening of soft structures over the joint, ganglionic cyst, congenitally wide head of the first metatarsal, hallux valgus, hallux rigidus, proliferation of the dorsum of the first metatarsophalangeal joint (dorsal bunion); and any proliferative changes secondary to arthritides. When applied to the fifth metatarsal head (tailor's bunion), *bunion* has been misapplied to connote lateral bending of head, congenitally wide head, and chronic thickening of soft structures over the head. As a corollary, *bunionectomy* is an equally unscientific term It must be recognized that enlargement of the tibial side of the great toe joint, generally described as *bunion,* may be due to a ganglion, to congenitally wide head, to hallux valgus, or to hallux valgus with ganglion (Fig 309), and other unusual enlargements of this joint Those and related disorders are herewith individually considered

Congenitally Wide First Metatarsal Head

A congenitally wide first metatarsal head (Fig 310) often becomes a pressure area on the medial side of the great toe joint Whether or not it is associated with hallux valgus, it usually becomes a problem in its own right The wide head of its medial side becomes the most prominent point on the inner surface of the foot, creating a fulcrum for pressure by footwear and resulting in a chronic inflammatory process of the synovial structures over the area The inflammation produces a further thickening of the synovia and enlargement of the prominence

Symptoms.—The symptoms are chronic pain and swelling over the medial side of the first metatarsal head. A shoe cut out over this area gives relief, but treatment requires amputation of the tibial condylar process of the first metatarsal head



A B C D E

F

Fig 309 —*A*, Normal great toe joint *B*, Congenitally wide metatarsal head *C*, Ganglionic cyst on tibial side of first metatarsal head *D*, Hallux valgus *E*, Hallux valgus and ganglionic cyst *F*, Massive ganglion and hallux valgus

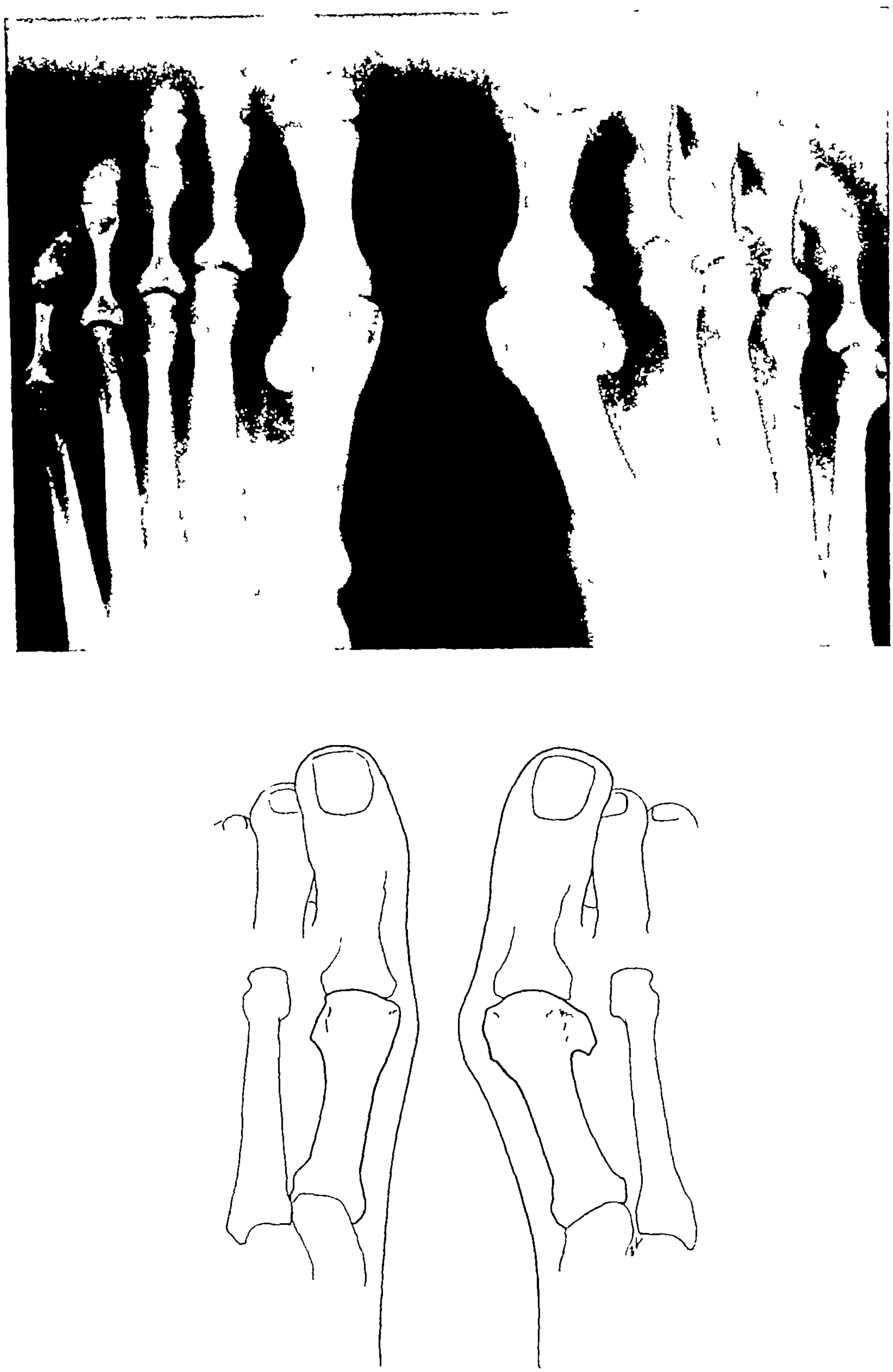


Fig 310 – Left, Normal-shaped metatarsal head Right, Congenitally wide metatarsal head in same person

Technique for Amputation.—Complications after the procedure herewith described are rare

1 Make a semilunar incision with its vertex extending dorsally from the middle of the tibial side of the first proximal phalanx to a point about 3 cm proximal to the head on the tibial side of the first metatarsal

2 Dissect and free the skin flap formed on the medial side of the joint to expose the tibial side of the great toe joint

3 Excise all excess and hypertrophied bursal tissue

4 Make a longitudinal incision in the mediodorsal aspect of the joint capsule, dissecting it as well and denuding it from the base of the proximal phalanx and head of the first metatarsal

5 Retract the capsular flap with the skin flap, exposing the tibial condyle of the first metatarsal head

6 By severing the metatarsophalangeal interarticular ligament, deliver the head of the metatarsal dorsomedially out of the wound by means of a periosteal or Chandler elevator

7 The condylar ridge on the articular surface of the metatarsal head makes an ideal landmark for removal of the tibial condylar process by means of a straight nasal saw, then round the sharp margins with a Joseph's nasal rasp

8 Instill from 5 to 10 mg of hydrocortisone into the joint

9 Suture the capsule and skin in layers and apply a compression bandage

Ambulation may begin in twenty-four hours Full use of the joint is usually possible in two or three weeks

Hallux Valgus

Hallux valgus is a static partial dislocation or subluxation of the great toe to a lateral or valgus attitude (Haines and McDougall, 1954, Joseph, 1954) It involves all the osseous and soft tissue structures comprising and surrounding the first metatarsophalangeal articulation It is characterized by valgus (abduction) of the great toe and varus (adduction) of the first metatarsal This deformity is by far the one most commonly classified as *bunion* It is the most disabling and most complex problem among morbidities of the great toe joint

Etiology.—The etiology of hallux valgus has not been established Erroneous concepts have resulted from the search for a single cause Recorded causes are often contradictory Mayo (1908, 1920) believed that the longer the length of the first metatarsal and great toe in relation to the other toes, the likelier is this malady to occur Morton, on the other hand, in 1935 was of the opinion that the short first metatarsal is etiologic in hallux valgus McElvenny (1944) and Truslow (1925) emphasized heredity as the etiologic factor Robinson (1927) observed that the movement of the sesamoid into the first metatarsal interspace is the direct cause of the deformity He minimized the effect of a faulty shoe Kaplan (1955) regarded certain variations in the action of the tibialis posticus muscle as causative Hardy and Clapham (1951), Truslow (1925), and Hawkins and his associates (1945) saw a high degree of correlation between hallux valgus and wide intermetatarsal angle (metatarsus primus varus) Galland and Jordan

(1938) regarded hallux valgus as an outcome of the valgus position of the heel and tarsus McBride (1935) attributes the deformity to faulty muscular equilibrium brought on by improper shoes. Kleinberg (1932) considers the primary cause that of the adducted first metatarsal and the unusual obliquity of the first tarsometatarsal joint Durman (1956) stresses metatarsus primus varus in causation

Predisposing Factors.—Anatomic variants from the normal predispose to hallux valgus It is in this anatomic sense that the deformity may be considered hereditary We inherit all our anatomic variations The first metatarsophalangeal joint is the most complex structure of the foot The complexity is accentuated by concentration of the functions of the forefoot on the medial border of the foot, so that any anatomic deviation of the medial border may influence causation of hallux valgus Even the muscles of the leg and their variations may be causative

The main congenital or hereditary anatomic predisposing conditions are as follows (1) the convexity of the head of the first metatarsal, (2) the angular relationship of the base of the first metatarsal to the first cuneiform, (3) anatomic variations of the adductor and abductor hallucis, (4) relative articular surfaces of the dorsum of the sesamoids to the plantar surface of the head of the first metatarsal, (5) a first metatarsal longer than the second metatarsal, and (6) hereditary tendency to flaccid ligaments

Convexity of Head of First Metatarsal (Figs 311 and 312) The more convex the head, the likelier is the proximal phalanx to rotate lateralward on it.

Angular Relationship of Base of First Metatarsal to First Cuneiform The further the joint deviates from a right angle, which also increases the intermetatarsal angle at the base of the first and second metatarsals—metatarsus primus varus (Fig 313)—the likelier is the deformity to develop Durman (1956) observed that the normal intermetatarsal angle at the base of the first and second

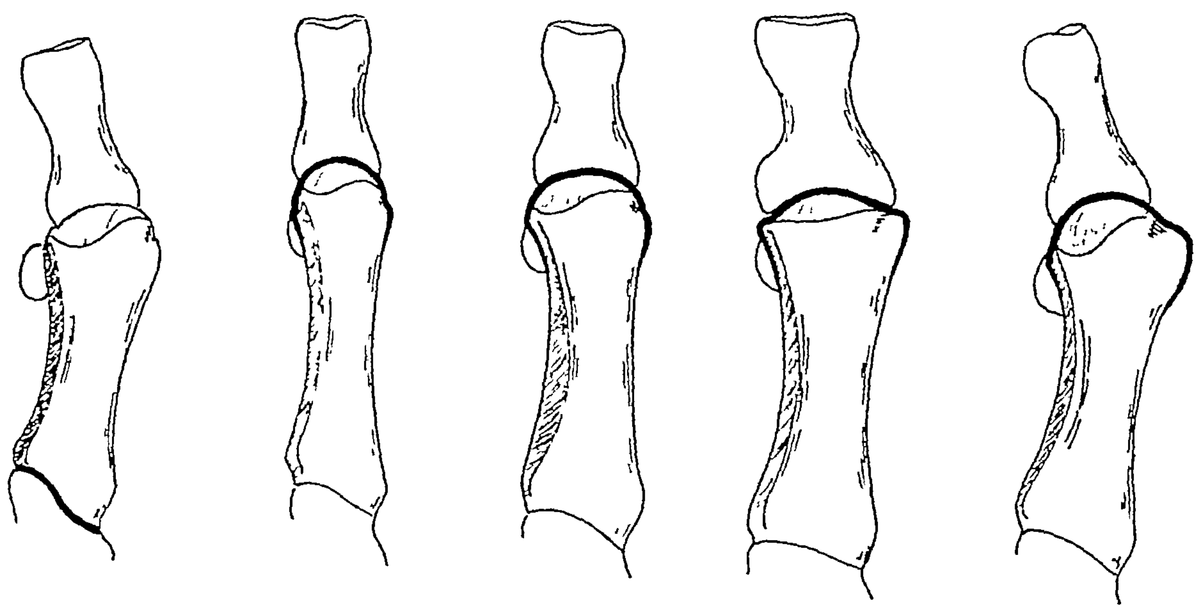
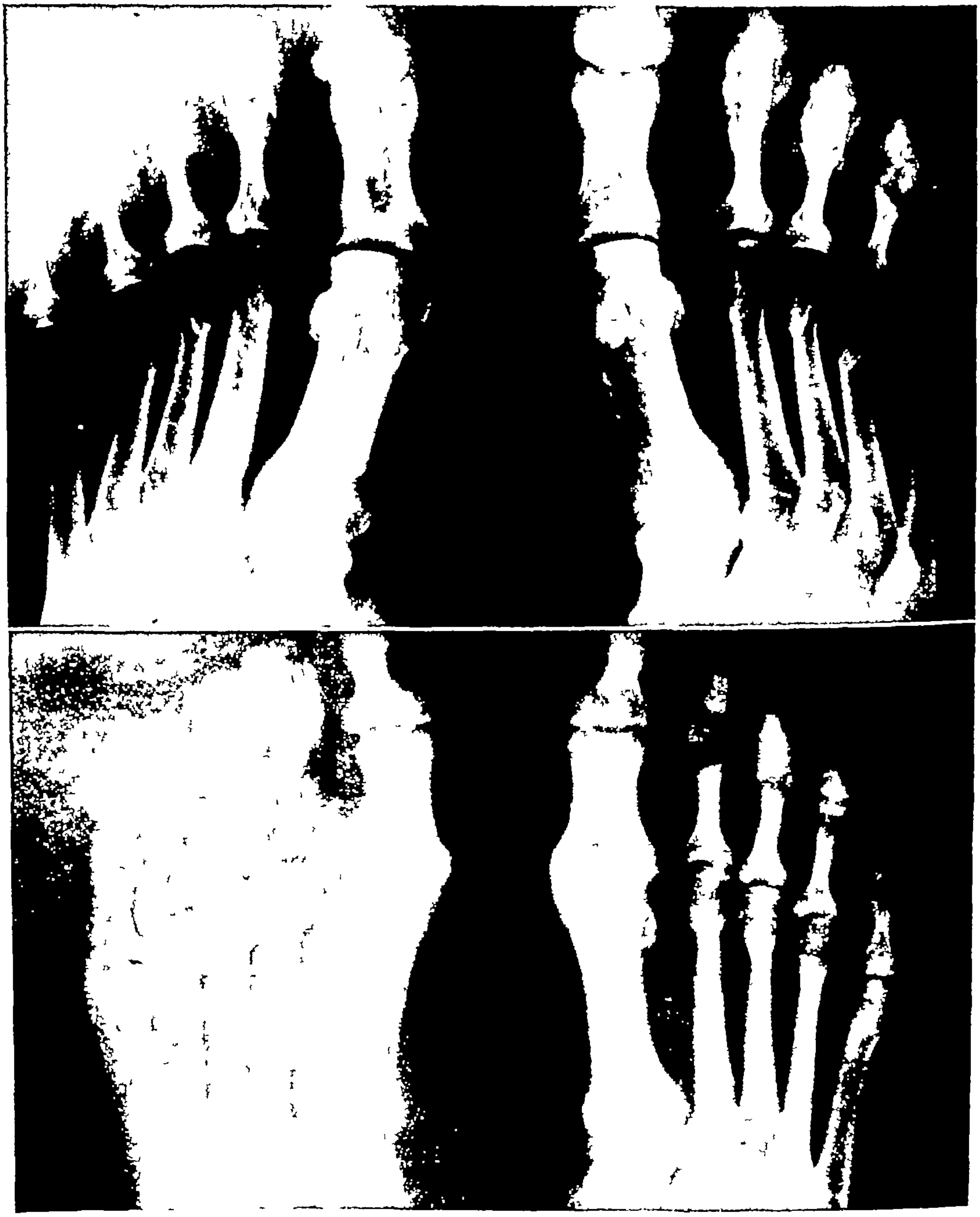


Fig 311 —Some variations in shape of first metatarsal head, contributing to deformity of first metatarsophalangeal joint

A

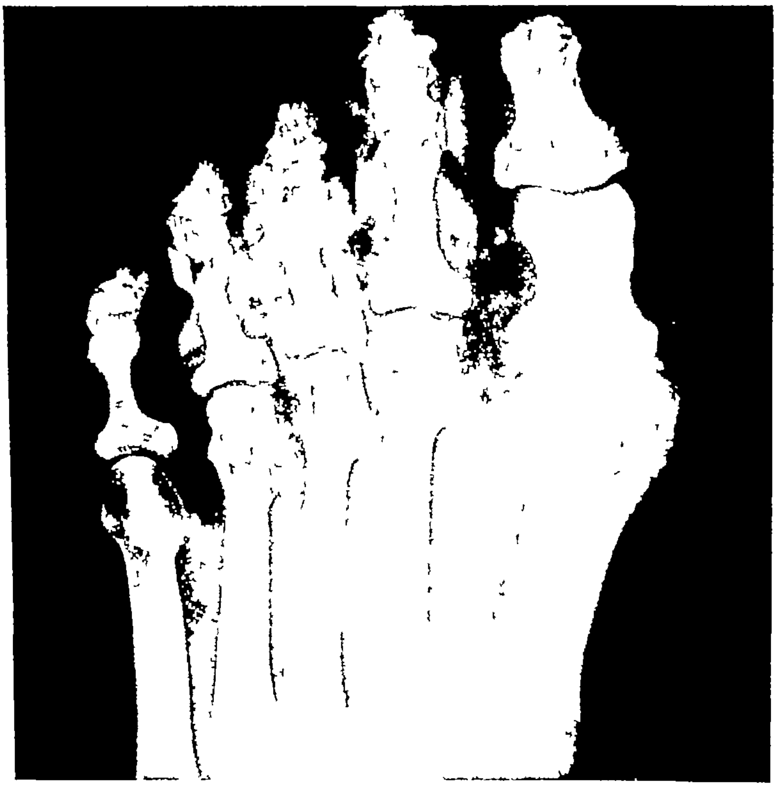


B

Fig 312.—*A*, Flat type metatarsal head, resists hallux valgus deformity *B* and *C*, Peculiar shape of metatarsal heads resists movements of great toe into valgus position

metatarsals is between 5 and 8 degrees, and an increase in the angle (metatarsus primus varus) is the underlying cause of hallux valgus

Pull of Adductor Hallucis Normally, the two opposing muscles at the sides of the base of the proximal phalanx, namely, the adductor hallucis and abductor hallucis, maintain the forward alignment of the great toe on the metatarsal shaft in equilibrium There are no muscle insertions in or around the metatarsal head, however, the base of the proximal phalanx is the site of insertion of those two opposing muscles as well as of the flexor and extensor hallucis brevis As a result, the metatarsal head becomes a fulcrum, the great toe, a lever The adductor hallucis is normally far more powerful than the abductor It was intended to



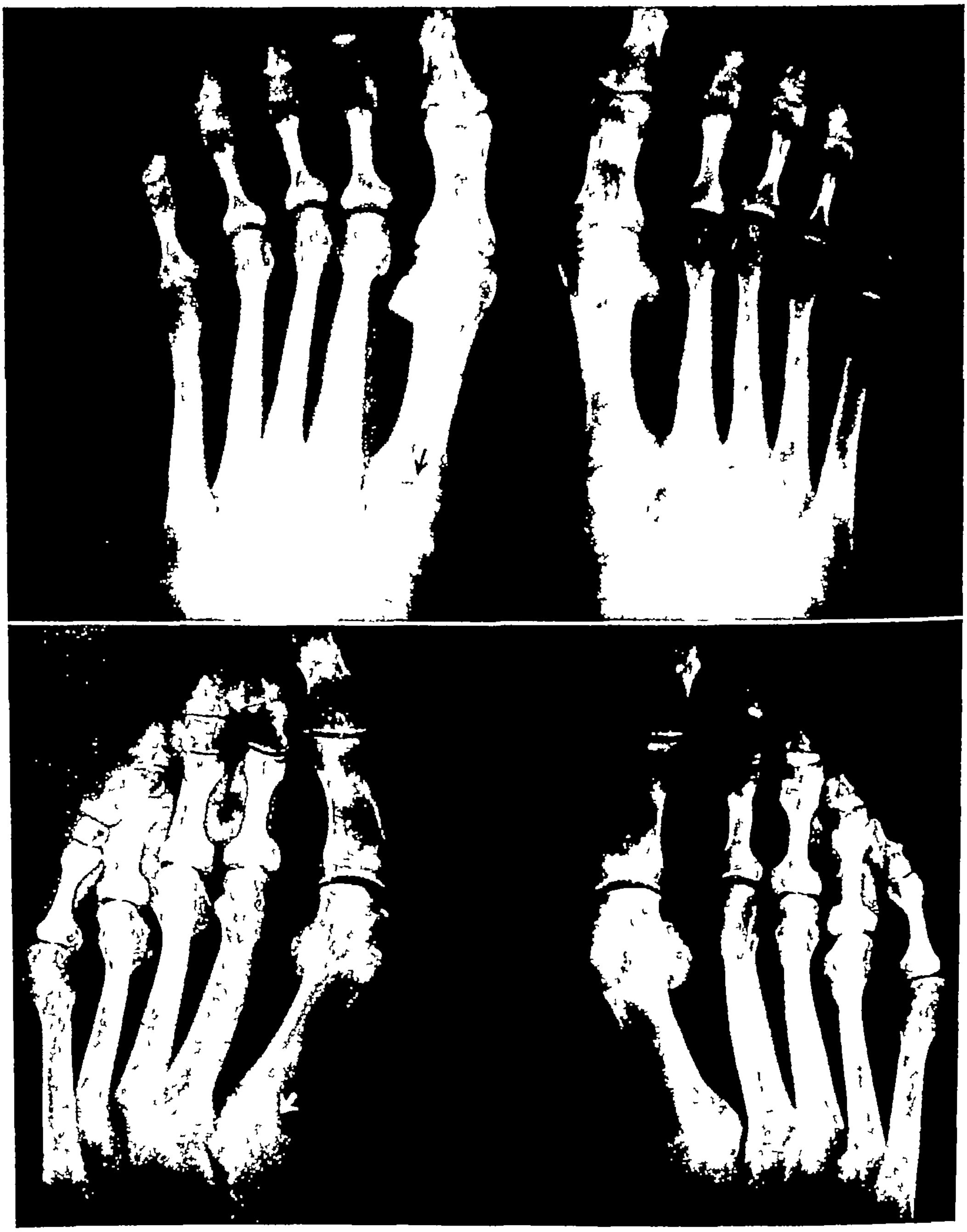
C

Fig 312 (cont'd) —For legend see opposite page

grip and hold objects, whereas the function of the abductor is only to abduct the toe When, however, there are anatomic variants, for example, an oval-shaped metatarsal head or an angulated first metatarsal-first cuneiform articulation or perhaps both, then the adductor tends to pull the base of the first proximal phalanx of the head off the first metatarsal (Fig 314)

Relation of Articular Surfaces of Dorsum of Sesamoids to Plantar Surface of Head of First Metatarsal The tibial sesamoid is convex transversely This convexity fits into a sulcus on the plantar surface of the first metatarsal head formed by a tibial lip and middle ridge The shallower the sulcus for the articulation of the tibial sesamoid, the more readily does the first metatarsal deviate in a varus position (Fig 315)

A



B

Fig 313 —*A*, Base of first metatarsal articulates with first cuneiform at a right angle, resists hallux valgus *B*, Base of first metatarsal articulates with first cuneiform in obtuse angle Conducive to hallux valgus

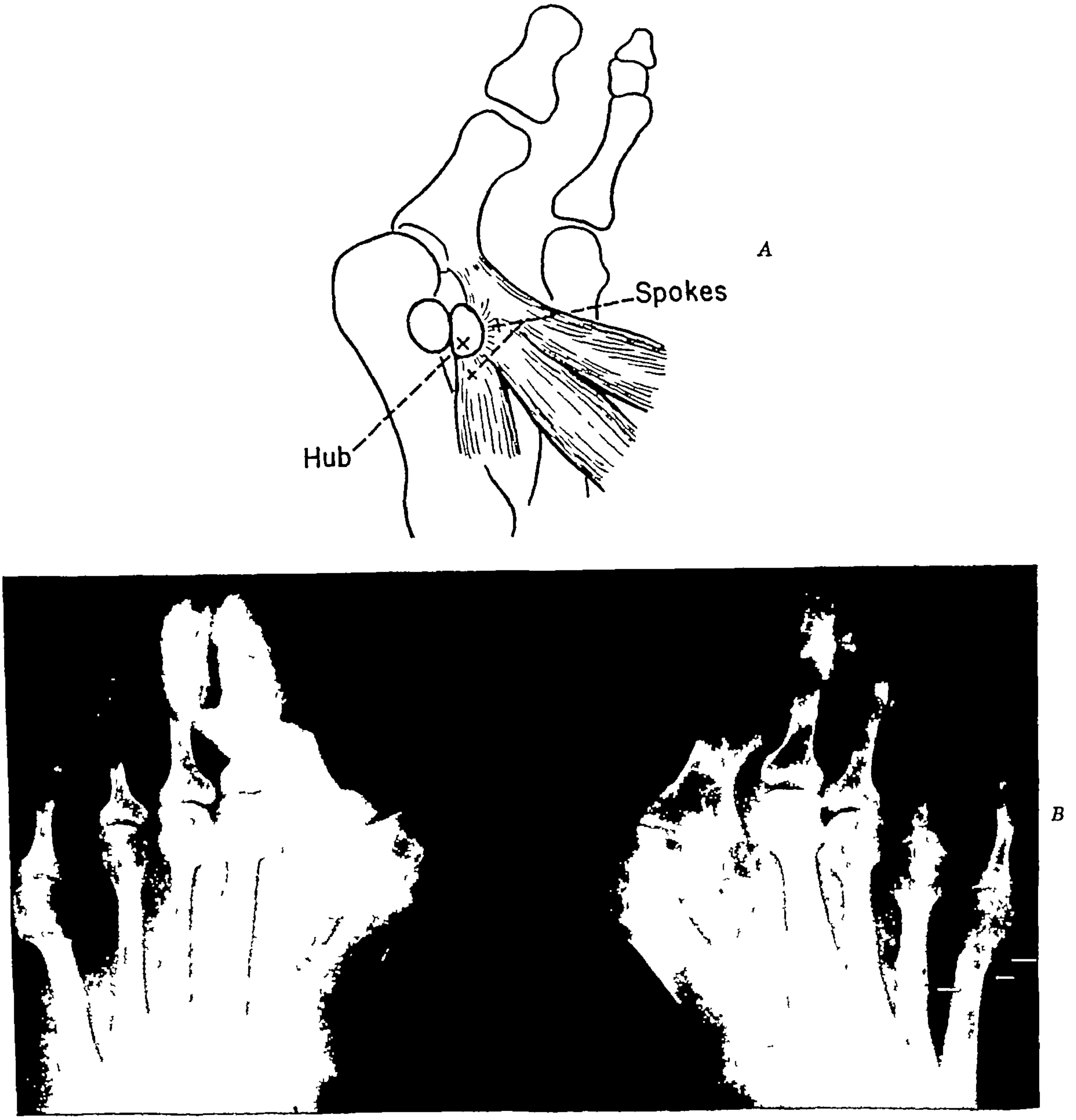


Fig 314 —*A*, Conjoined tendon of adductor hallucis and fibular portion of flexor hallucis brevis tendon pull proximal phalanx off first metatarsal head *B*, Actual case in which adductor hallucis had pulled proximal phalanx off first metatarsal head

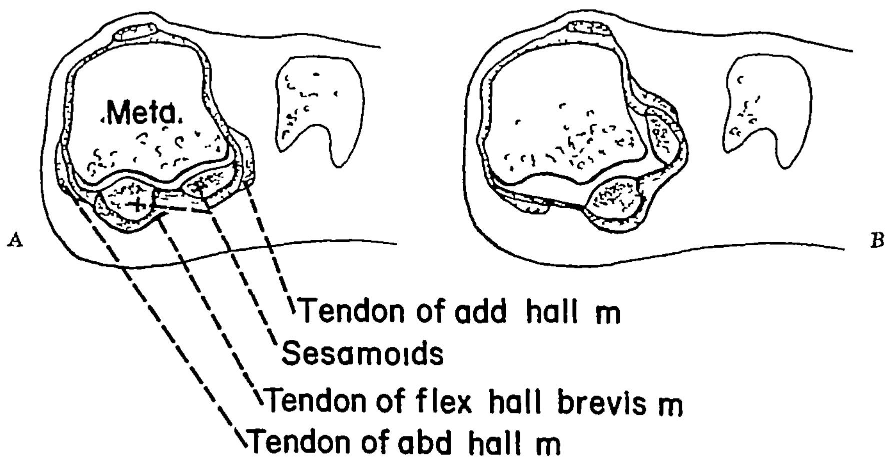


Fig 315 —Cross section of first metatarsal head and sesamoids *A*, Normal relationship between metatarsal head and sesamoids and tendons coursing in area *B*, Hallux valgus Note distortion of all structures around metatarsal head

Fig 316

Fig 317

Fig 316 —First metatarsal longer than second metatarsal Base of first proximal phalanx unstable

Fig 317 —Severe hallux valgus resulting from amputation of second toe

First Metatarsal Longer Than Second Metatarsal Because the base of the first proximal phalanx, into which the powerful adductor hallucis is inserted, has no anchorage such as is afforded by the head of the usually longer second metatarsal (Fig 316), hallux valgus may be induced For that reason, amputation of the second toe is ill advised If the patient did not previously have hallux valgus, the condition will develop as a result of amputation (Fig 317) If the deformity is already present, it will be aggravated

Hereditary Tendency to Flaccid Ligaments When flaccid ligaments are hereditary, a flaccid musculature is often concomitant, and the two may induce hallux valgus Flaccidity may be restricted to the first segment and thus present the so-called hypermobile first segment When the whole forefoot has loose ligamentous structures, particularly intermetatarsal ligaments, all the metatarsals fan, and splayfoot is present Such flaccidity yields the joint to deformity Mechanical forces, such as ill-fitting shoes, easily mold the foot having flaccid ligaments Malalignment of the osseous structures ensues, secondarily causing changes in the soft tissue and in the articulating surfaces of bone Wolff's Law (page 346) and Davis' Law (page 53) apply in explaining how in time the malaligned structures become permanently fixed

Inciting Factors.—The most direct inciting factor is that of improper foot covering The short or pointed shoe holds the great toe in a position of valgus Short tight stockings or socks have somewhat the same effect The boxing of the short shoe is usually of a rigid material, so that it is not easily pushed out of shape It is firmly attached to the sole, preventing any demonstrable over-all increase in length The first metatarsophalangeal joint inevitably buckles the metatarsal into a pronounced varus position and the great toe into an extreme valgus attitude The pliable leather of the shoe upper over the medial side of the first metatarsophalangeal joint offers some resistance but is soon overcome by the unremitting line of force of the first metatarsal head, hence, the characteristic bulging of the shoe over the first metatarsal head when this deformity is present

Contributory Factors.—Flatfoot, long periods of standing, and predisposing anatomic variations represent contributory factors

Flatfoot and Occupation —Flatfoot is frequently associated with hallux valgus and is often a contributory factor in the cause or is a forerunner of the cause An occupation requiring long periods of standing may be directly contributory

Congenital —Congenital hallux valgus may be bilateral or unilateral All or many of the anatomic variations predisposing to acquired hallux valgus will be present in the congenital condition to an extreme degree Valgus deformity of the interphalangeal joint may result from an ill-shaped head of the proximal phalanx

Pathologic Anatomy.—The proximal and distal phalanx are deviated toward the fibular side The first metatarsal head is deviated toward the tibial side of the foot The capsule on the tibial side of the joint is stretched, and the capsule on the fibular side of the joint is contracted The adductor hallucis is lengthened The abductor hallucis tendon is elongated The tendons of the extensor hallucis longus and flexor hallucis longus are shortened and act as bowstrings The tibial sesamoid is in the place normally occupied by the fibular sesamoid The fibular sesamoid is in the first metatarsal interspace The sesamoids are really not dis-

placed. It is the metatarsal head that moves off of the sesamoids The fibular sesamoid becomes the hub of the deformity The conjoined tendon of the adductor hallucis and the fibular half of the flexor hallucis brevis tendon act as spokes of a wheel, holding the fibular sesamoid at the hub (Fig 318). That is why during surgical procedures when this sesamoid is excised, it is immediately possible to reduce the deformity manually

According to comparative studies* of 1,000 roentgenograms of hallux valgus with 1,000 roentgenograms of feet without hallux valgus, exostotic changes were not more in evidence than in normal feet There are no osseous proliferative changes in the tibial condyle of the head of the first metatarsal, therefore, *exostosis*, a consistent misnomer, does not exist (Bernard, 1930, Brookes, 1935, Kreuscher and Kelikian, 1935, Levine, 1938, Stein, 1938)

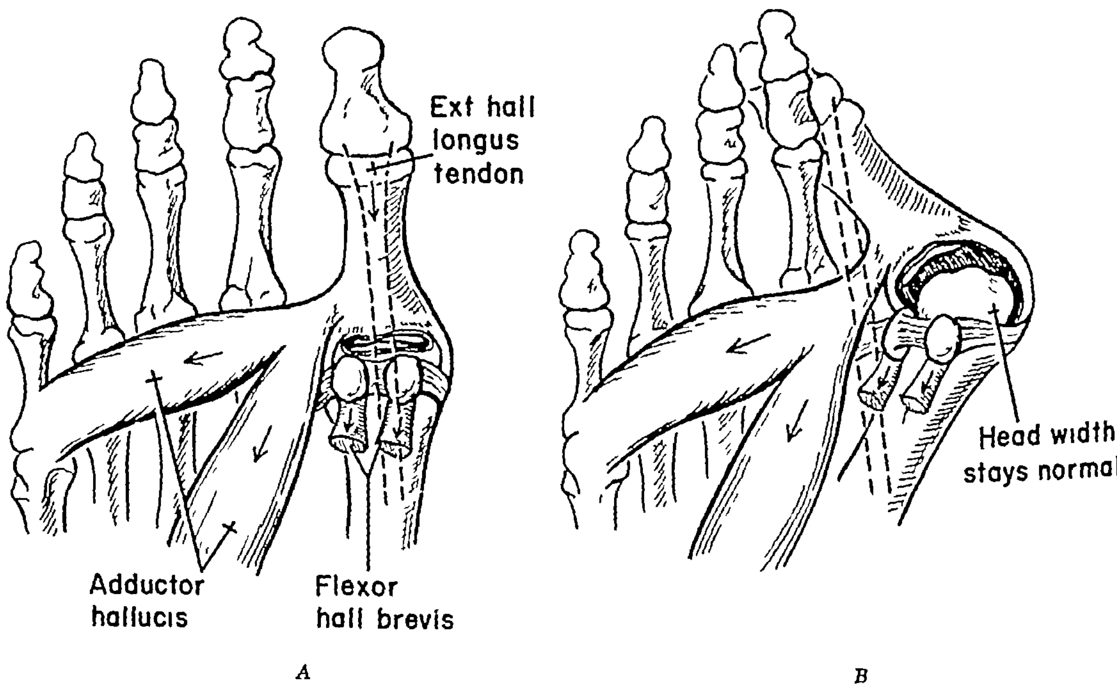


Fig 318 —Pathologic anatomy of hallux valgus *A*, Normal anatomic ensemble of first metatarsophalangeal joint. *B*, Hallux valgus with its distortion of all the anatomic structures around first metatarsophalangeal joint

Treatment.—Although reports are fragmentary regarding the etiology of hallux valgus, reports of operative procedures for its correction (Fig 319) are numerous (Galland and Jordan, 1938)

Simple and Modified Condylectomies.—Simple condylectomy (exostosectomy) of the tibial side of the first metatarsal head is one of the commonest procedures advocated, because it is simple and is seldom disabling It only produces disability when performed in cases of severe dislocation of the great toe joint or when too much of the tibial side of the head of the first metatarsal has been removed McElvenny and Thompson (1940) and Levine (1938) also removed

*Personal studies by the author

the tibial condylar lip of the base of the first proximal phalanx, however, that is of value only when the protuberance is due entirely to an unusually wide first metatarsal head It is of little or no value when there is extreme hallux valgus, it may even worsen the deformity

Stanley and Breck (1935) reported the results in 211 cases in which they removed the tibial condylar side of the head of the first metatarsal "exostosis" through a unique web incision (Fig 320)

Arthrodesis.—Girdlestone and Spooner (1937) and Joplin (1950) considered splayfoot a forerunner of hallux valgus Girdlestone and Spooner aimed to correct the condition by arthrodesis of the base of the proximal phalanx to the head of the first metatarsal (Fig 321), thereby making use of the adductor hallucis to pull the metatarsal heads together

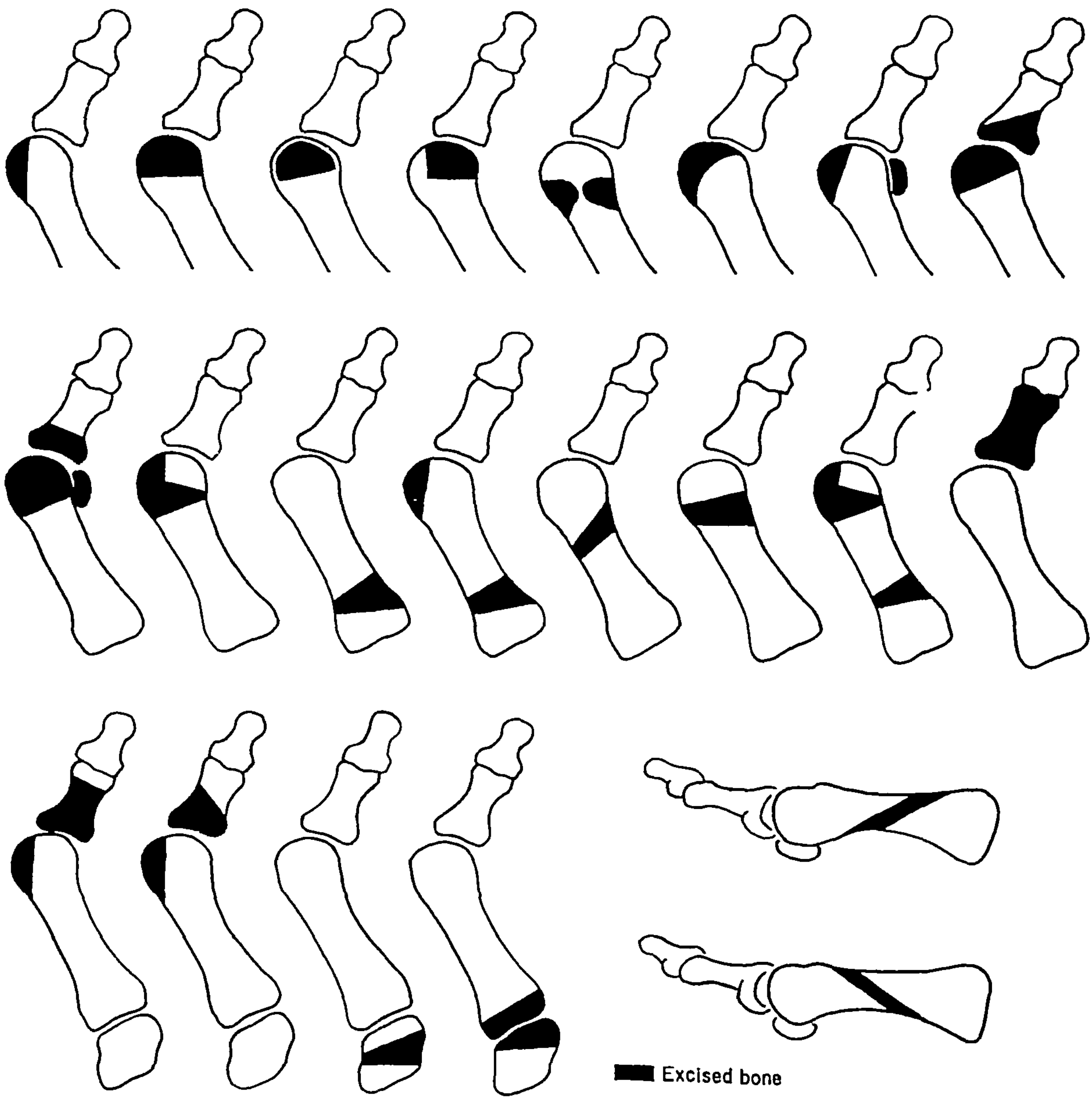


Fig 319 —Some methods of excisions of bone for hallux valgus

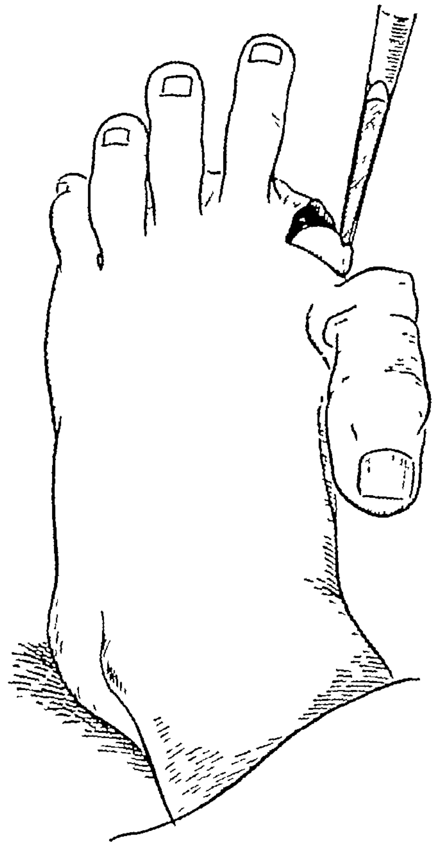


Fig 320 —Stanley and Breck's technique for hallux valgus

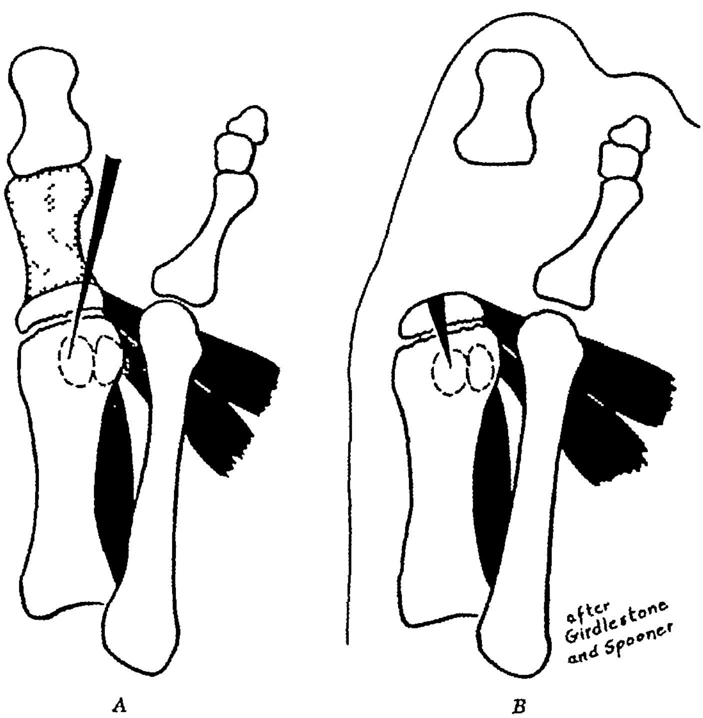


Fig 321 —Girdlestone and Spooner's technique for hallux valgus A, Distal seven-eighths of proximal phalanx excised B, Base of proximal phalanx anchored to head of first metatarsal

Plantar Sling.—Joplin made a transverse plantar sling of the extensor longus digitorum tendon from the fifth toe (Fig 322) and brought under the metatarsal arch

Condylectomy Combined With Step-Down Osteotomy.—Hawkins and his associates (1945), in addition to removing the tibial condylar side of the first metatarsal head, perform a step-down osteotomy on the first metatarsal shaft (Fig 323) They report excellent results in a large series of cases

Subcapital Amputation.—Hueter was one of the first (1887) to advise subcapital amputation of the first metatarsal head The procedure was later modified by Mayo (1908) and Soresi (1931), among others It is a commonly practiced procedure

In all first metatarsal subcapital amputations, the base of the proximal phalanx moves by the pull of the flexor hallucis brevis and abductor hallucis into the space formerly occupied by the head, thereby straightening the toe and producing excellent cosmetic results However, the functional results are limited,

Fig 322 —Joplin's sling technique for hallux valgus Extensor tendon of fifth digit threaded under metatarsal heads, with tendon of adductor hallucis threaded through a drill hole in first metatarsal head, sutured to capsule on medial side (Courtesy American Academy of Orthopaedic Surgeons, Inc)

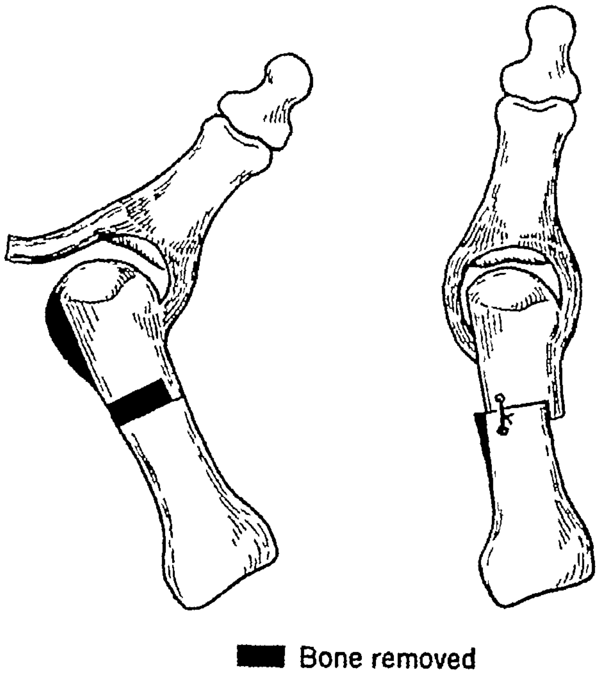
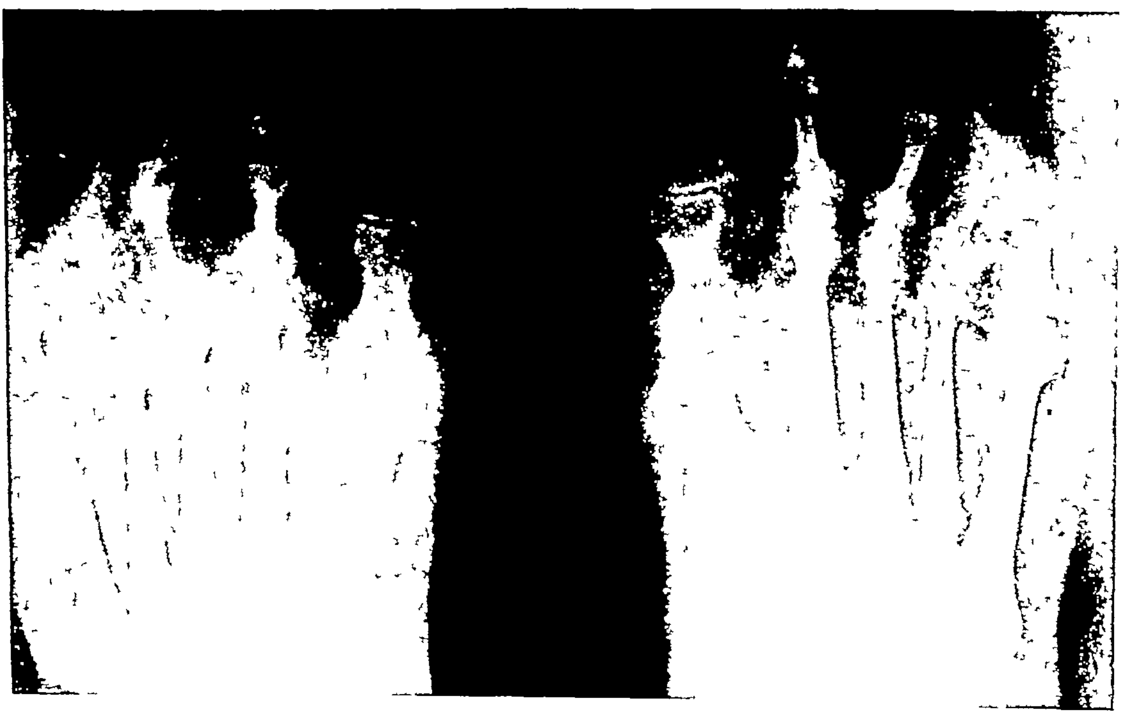


Fig 323 —Hawkins' step-down osteotomy of first metatarsal shaft for hallux valgus

A

Fig 324 —A-C, End results of subcapital amputation of first metatarsal head for hallux valgus

B



C

Fig 324 (cont'd) –For legend see opposite page

because the weight-bearing surface of the first metatarsal head has been destroyed. A personal review of one hundred cases of severe disability resulting from subcapital amputation emphasizes that function of the first metatarsal is as important as the weight bearing of all the other four metatarsals, thus, the subcapital amputation is deplored

The distal half of the shaft of the first metatarsal receives its blood supply from the epiphyseal head, therefore, the danger is always likely of destroying the blood supply by subcapital amputation and thus leading to atrophy of the distal half of the shaft (Fig 324) False ankylosis of the first metatarsophalangeal joint may ensue if the head is not rounded during the procedure for subcapital amputation When the head is angulated during amputation, the articular surface of the head is left pinpointed, and a gouging action is produced into the base of the proximal phalanx (Fig 325)

Fig 325 —Pointed articulation of head of first metatarsal with base of proximal phalanx, after subcapital amputation Patient was in constant pain when walking

Amputation of Proximal Half of Proximal Phalanx—Keller described his operation for amputating the proximal half of the first proximal phalanx for hallux valgus correction in 1904, later (1912) he reported refinements His procedure has acquired support, notably by Brandes (1929), Cleveland and Winant (1950), Schein (1940), and Galland and Jordan (1938) It is probably the most widely used procedure in the United States

Keller Technique—An elliptical incision, having its base dorsally, is made over the great toe joint A U-shaped incision with its base about 1 cm distal to the base of the proximal phalanx is made in the tibial capsule of the great toe joint. The flap is dissected distally to expose the medial surface of the head of the

first metatarsal and the base of the proximal phalanx The medial condyle of the first metatarsal is amputated The attachments to the base of the proximal phalanx are freed About 1 or 1 5 cm or the proximal third of the base of the proximal phalanx is amputated and smoothed with a rasp. The toe is brought into alignment and the U flap sutured to the periosteum just behind the tibial side of the head of the first metatarsal This is done under mild tension, with the toe slightly overcorrected The skin is sutured.

Evaluation The Keller method gives excellent cosmetic results, however, the great toe is always shortened, its function in walking is diminished and in some cases the toe becomes a useless appendage That happens because the important action of the great toe is controlled by the intrinsic muscles that are inserted into the base of the proximal phalanx, and that part of the bone is amputated, thereby destroying the function of these muscles

Fig 326 —Sesamoids receded to middle of first metatarsal shaft because flexor hallucis brevis insertion had been destroyed during Keller procedure

The sesamoids usually recede proximally (Figs 326 and 327), because the flexor hallucis brevis insertion has been destroyed and the weight-bearing function of the sesamoids has been lost Hammered great toes may also follow this process as a result of the severance of the flexor hallucis brevis which reduces the counteraction against the extensor hallucis longus so as to dorsiflex and produce the disabling hammertoe deformity

Capsulotomy —Silver (1923) was one of the first to call attention to muscle imbalance, especially imbalance of the intrinsic muscles, in the etiology of hallux valgus He considered those muscles basic in the pathologic alterations producing the deformity, consequently, the operation should have as its object the cor-

because the weight-bearing surface of the first metatarsal head has been destroyed A personal review of one hundred cases of severe disability resulting from subcapital amputation emphasizes that function of the first metatarsal is as important as the weight bearing of all the other four metatarsals, thus, the subcapital amputation is deplored

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rection of the pathologic disorder without mutilating a joint unscientifically in order to overcome deformity of the joint

Silver's Technique for Reduction of Hallux Valgus—A semilunar incision is made over the medial surface of the great toe joint, with its vertex dorsally. A Y-shaped incision is made in the capsule to form three flaps one distal, one dorsal, and one plantar. The flaps are dissected so that the head of the metatarsal is exposed While the great toe is plantar-flexed, a tenotome is inserted just above the capsule of the plantar joint, and a longitudinal incision is made on the fibular side of the capsule With the toe dorsiflexed, a longitudinal incision is made in the dorsal margin of the capsule on the fibular side The toe is strongly adducted A vertical incision is made on the fibular capsule between the two longitudinal incisions This capsulotomy releases the tension on the fibular side of the joint The toe is overcorrected and the distal flap on the medial side is sutured to the periosteum just behind the head of the first metatarsal The dorsal and plantar flaps are sutured over the distal flap

McBride (1928, 1935, 1954) supported Silver regarding the mechanical influence on deformity by "contraction of the muscles whose conjoined tendon inserts into the base of the external aspect of the first phalanx of the big toe" He further called attention to the usual displacement of the fibular sesamoid into the space between the first and second metatarsal heads

McBride's Technique—Through an incision over the first metatarsal interspace the conjoined tendon of the adductor hallucis is detached from its insertion, and the fibular sesamoid is excised The medial margin of the incision is retracted over the tibial side of the great toe joint, and the tibial side of the head of the first metatarsal is amputated The conjoined tendon of the adductor hallucis is tranferred into the dorsum of the head of the first metatarsal bone

Evaluation McBride was the first to suggest a procedure for the correction of hallux valgus which aimed at the correction of the underlying pathologic-anatomic disorder His procedure, although scientific and rewarding when properly performed, has not been favored It is too difficult to accomplish without training and practice

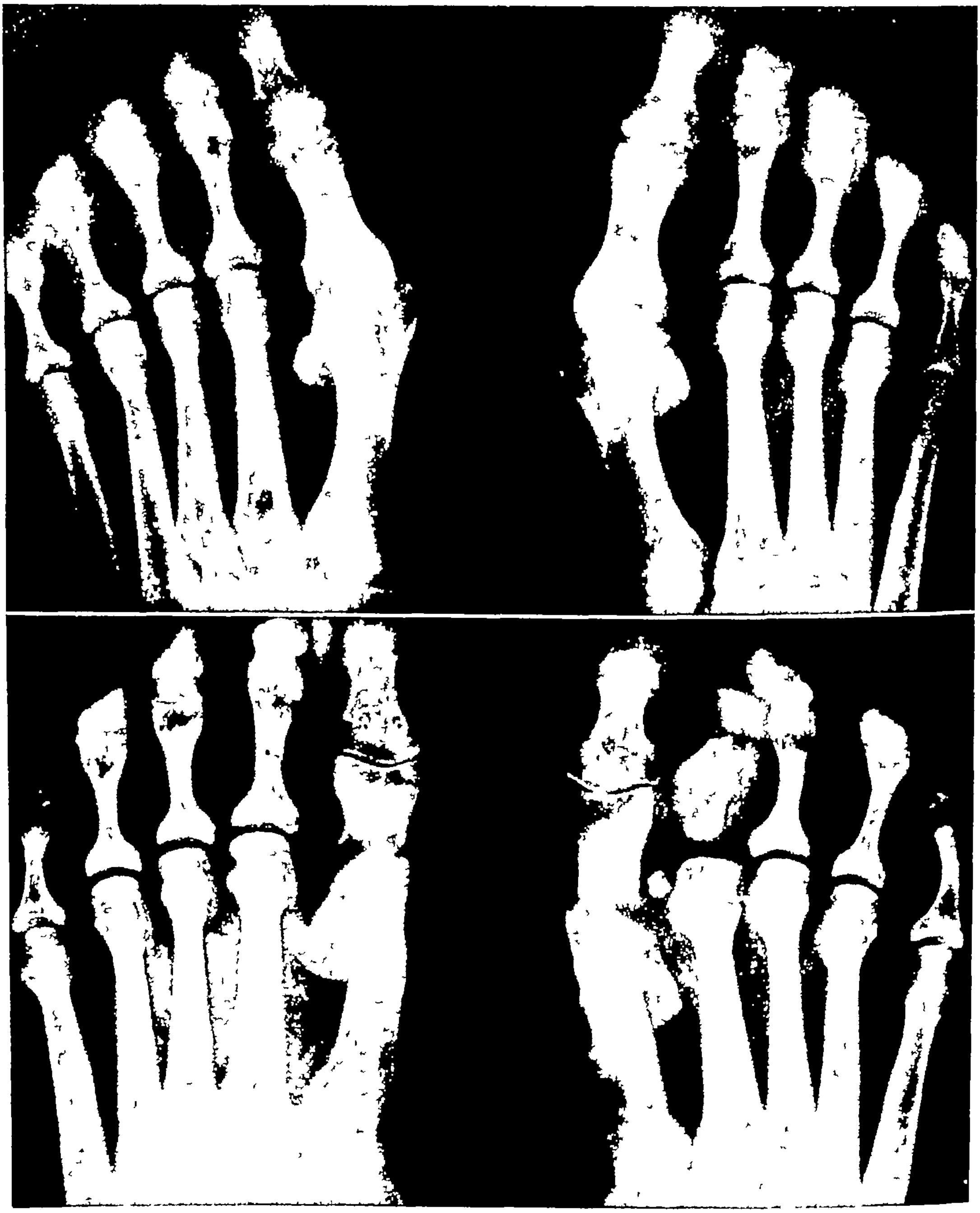
Recommended Procedure for Reduction of Hallux Valgus, DuVries' Modified McBride Technique—In a series of 1,200 cases of hallux valgus over a twenty-five-year span, reduction by means of a modified McBride procedure proved excellent in 90 per cent of the results, good in 8 per cent, and a failure in 2 per cent but without worsening of the condition

1 Under hemostasis by means of a rubber tourniquet or pneumatic cuff, make an incision over the first metatarsal interspace, extending from the web proximally and medialward, crossing the tendon of the extensor hallucis longus, and terminating at a point near the mediodorsal aspect of the base of the first metatarsal (Fig 328, *1*)

2 Retract the skin, and spread the first and second metatarsal heads by means of a Weitlander or mastoid retractor (Fig 328, *2*)

3 Carry the distal end of the incision downward in the interspace until the tendon of the adductor hallucis comes plainly into view The dorsal cutaneous branch of the superficial peroneal nerve lies in the center of the incision It is

A



B

Fig 327 —*A*, Mild hallux valgus *B*, After Keller procedure Patient was in constant pain because of roughening of proximal surface of what remained of proximal phalanx Amputation of base of right second proximal phalanx made that digit flail and unstable

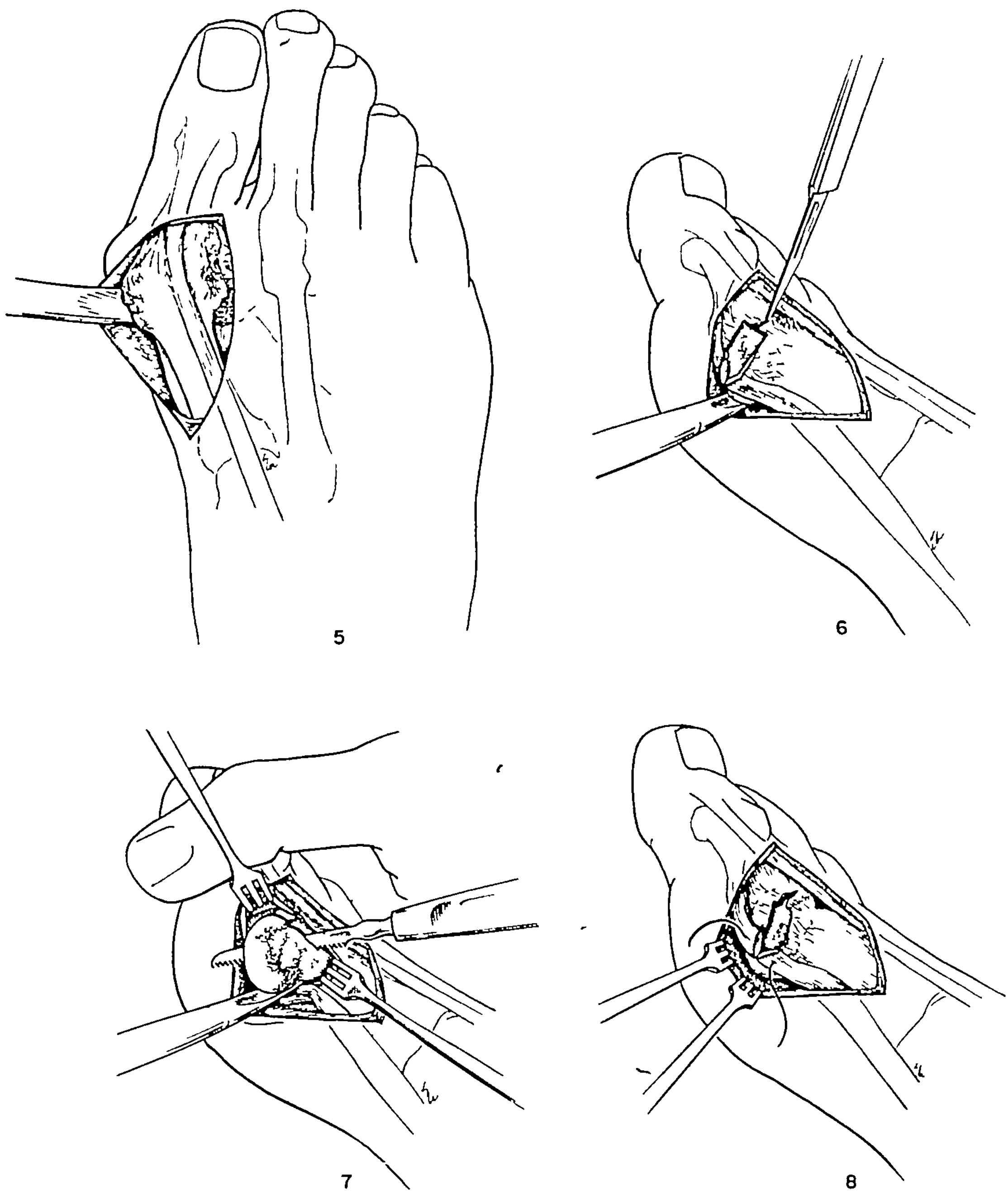
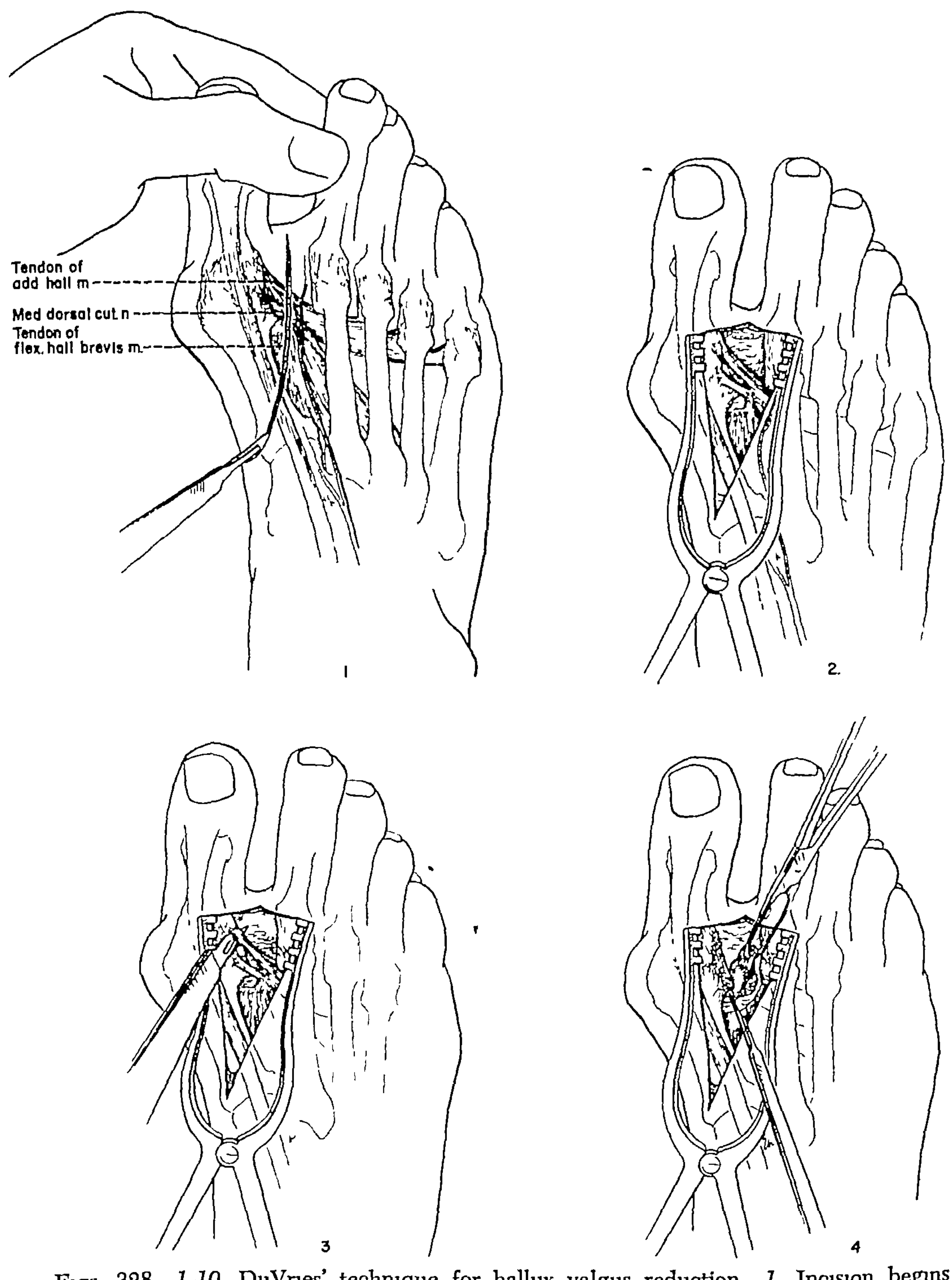


Fig 328 (cont'd) —For legend see opposite page

freed and retracted with the fibular margin of the incision If this nerve is severed, the patient will experience numbness of the great toe, in which case sensation either returns in three or four months or the patient ceases to be aware of anesthesia

4 Detach the adductor hallucis tendon from its insertion into the base of the proximal phalanx (Fig 328, 3)

5 Grasp the freed tendon by means of Allis forceps, and with slight pull on the tendon, bring the fibular sesamoid a little farther into the metatarsal interspace The stub of this tendon, which is conjoined with the tendon of the flexor hallucis brevis, can be used as a guide to excise the sesamoid (Fig 328, *4*)



Figs 328 —*1-10,* DuVries' technique for hallux valgus reduction *1,* Incision begins in first web and extends proximally and medialward, crossing extensor hallucis longus tendon, and ends over base of first metatarsal Mediodorsal cutaneous nerve in path of this incision *2,* Skin margins and cutaneous nerve retracted Incision carried downward in distal end of metatarsal interspace to expose insertion of adductor hallucis *3,* Detaching insertion of adductor hallucis *4,* Freed end of adductor hallucis grasped with *Allis forceps* This acts as guide for excision of fibular sesamoid *5,* Skin retracted over tibial side of first metatarsal head to expose tendon of abductor hallucis *6,* Vertical section about 0 5 cm excised from tibial capsule of first metatarsophalangeal joint and abductor hallucis tendon Dorsal margin of incision carried to extensor hallucis longus *7,* Tibial condylar process of first metatarsal head amputated with nasal saw *8* and *9,* Abductor hallucis tendon and capsule sutured in their shortened position *10,* Fascial structures between first and second metatarsophalangeal joints sutured together.

metatarsophalangeal joint to the capsule of the second metatarsophalangeal joint (Fig 328, *10*), this brings the two heads together

15 Instill 10 mg hydrocortisone acetate into first metatarsophalangeal joint

16 If the taut tendon of the extensor hallucis longus holds the great toe dorsiflexed, the tendon must be lengthened. The tourniquet placed over the calf instead of the thigh may cause a reflex spasm which may be mistaken for a contracted tendon

17 Suture the skin margins All sutures used are of nonabsorbable material except when the extensor longus hallucis has been lengthened, in which case its tendon sheath is sutured with plain catgut.

18 Apply a compression bandage while the great toe joint is immobilized in a normal position.

Fig 329 —Cut-out shoe, worn postoperatively after hallux valgus operation

19 Remove the tourniquet. Examine the foot for bleeding through the dressing If bleeding is present, apply an additional snug compression bandage

20 After twenty-four hours, release but do not remove the compression bandage Apply additional gauze to keep the foot snugly bandaged After three or four days, change the dressing completely The patient should be ambulatory at this stage but must wear cut-out shoes (Fig 329) In some cases, ambulation may not be advisable until the fifth or sixth day

21 Remove the sutures between the seventh and tenth day Apply a new snug bandage, with the great toe joint partly immobilized by a snug bandage and adhesive tape (Fig 330).

6 By sharp dissection against the body of the sesamoid, the sesamoid can be completely denuded and readily removed.

7. At this point the great toe joint can usually be aligned in a straight position In a few cases, some capsular fibers on the fibular side of the great toe joint may still hold the joint in deformity, if so, they must be sectioned

8. As soon as the great toe joint has been aligned, dissect and free the tibial margin of the incision and retract over the tibial side of the great toe joint by means of a periosteal or Chandler elevator placed between the skin and the plantar surface of the first metatarsophalangeal joint The capsule of the tibial side of the first metatarsophalangeal joint and the tendon of the adductor hallucis thus will have been completely exposed (Fig 328, 5)

9 Excise a vertical section about 0.5 cm wide from the tibial capsule of the first metatarsophalangeal joint, include the tendon of the abductor hallucis

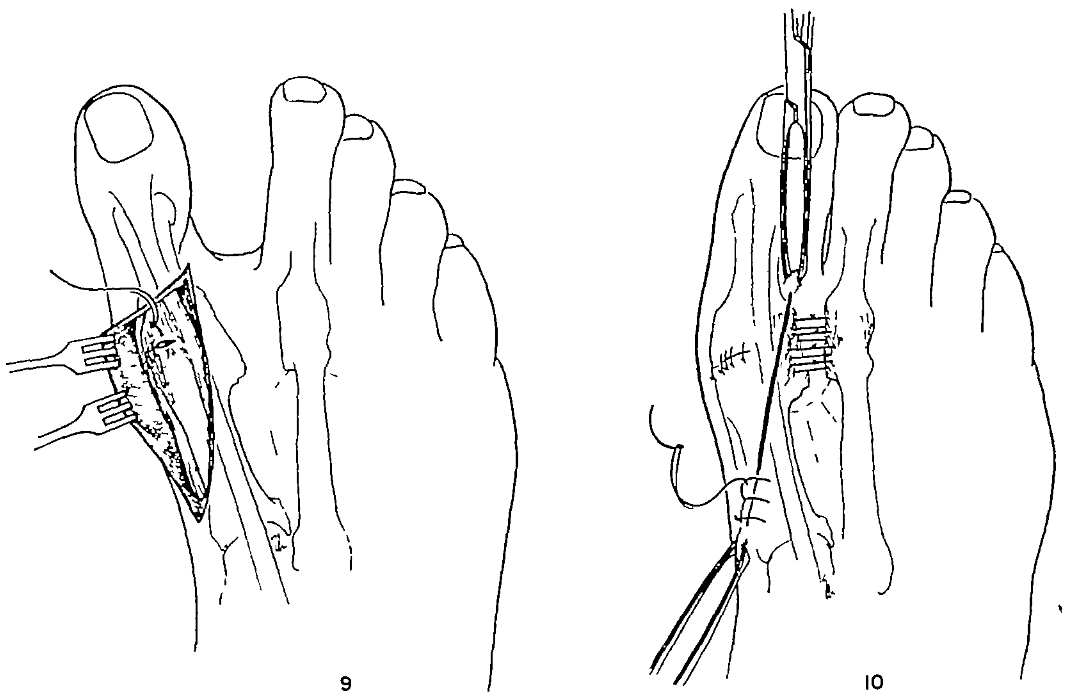


Fig 328 (cont'd) —For legend see page 382

10 After removal of this section, carry the vertical incision dorsally to about the medial margin of the extensor hallucis longus tendon (Fig 328, *6*)

11 Dissect and denude from the bone the distal and proximal margins of the capsule which will expose the tibial condylar process of the first metatarsal head

12 By means of a nasal saw, amputate the tibial condylar process of the first metatarsal head, beginning with the condylar groove on the articular surface of the head (Figs 318, *B*, and 328, *7*)

13 Round the head with a Joseph nasal rasp

14 Suture the capsule and tendon of the abductor hallucis in their shortened position (Fig 328, *8-9*) Suture the capsule on the fibular side of the first

22 Redress the wound every five to seven days Be sure that the great toe joint is maintained in a straight position for about two months, because until that time the tibial capsular structures which had been sutured are still fragile, any deviation of the toe or any pressure against the toe forces this capsule to give and elongate again Wedges of gauze or sponge rubber may be needed between the first and second toes in order to maintain the straight position (Fig 331) The patient wears the cut-out shoe during the two months, after which he is fitted with a new well-rounded toe shoe, appropriately designed The patient wears the new shoe for an additional three months or until he can wear other types of shoes comfortably Results are illustrated in Figs 332 and 333

A B

Fig 332—*A*, Hallux valgus and second metatarsophalangeal dislocation *B*, Three years postoperatively Note second metatarsophalangeal dislocation reduced without bone excision (See static second metatarsophalangeal dislocation, Fig 349)

Complications are uncommon When pyogenic infection is present, both local and general symptoms arise within seventy-two hours A broad-spectrum antibiotic is indicated at the least suggestion of infection It is administered under extreme vigilance until control is assured

Tinea infection is characterized by low-grade inflammation of the wound which may be mistaken for normal healing until its produces an avulsion of the incision when the sutures are removed The surgical area should be painted with a fungicide and the margins maintained with adhesive butterfly dressings Heal-

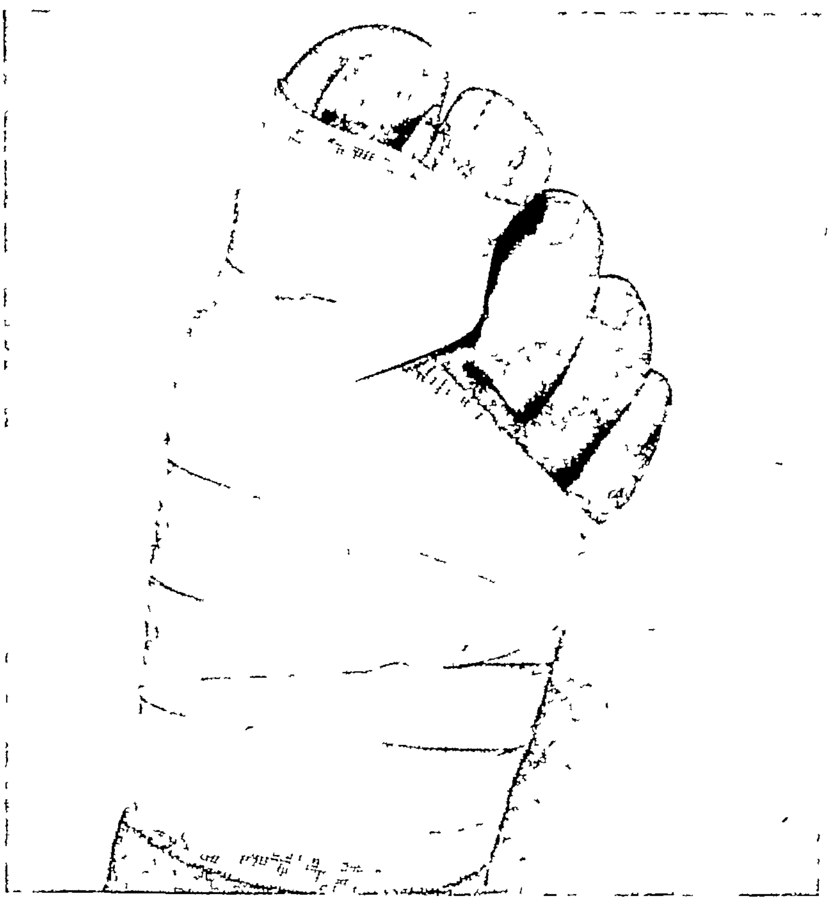


Fig 330 —Postoperative dressings for hallux valgus Second toe serves as splint. Sometimes third digit also included as splint

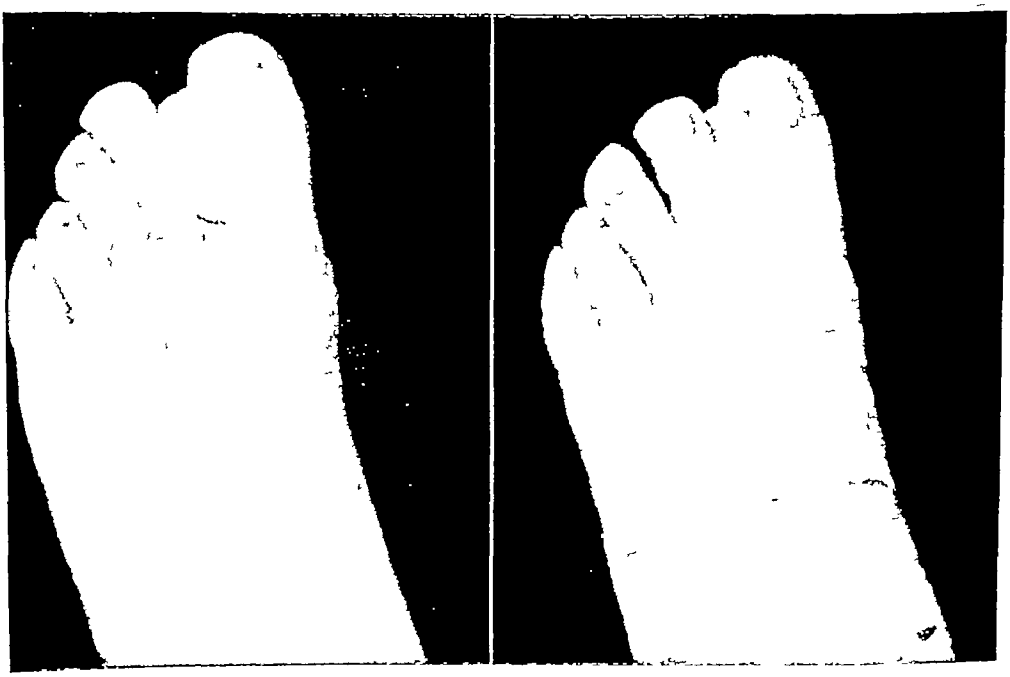
A  B

Fig 331 —*A*, Sponge rubber inserted between first and second toes *B*, Bandaging of foot with sponge rubber between first and second toes Dressing fits snugly over metatarsals but is neutral over digit This is important, so that when patient bears weight, sutures between first and second metatarsal heads will not spread apart

Hallux Varus

When the great toe is held or fixed in an inwardly bent position, the deformity is called *hallux varus* The defect may be congenital or postoperative Two surgical errors may cause the condition excessive shortening of the capsule and tendon of the abductor hallucis on the tibial side of the great toe joint (Fig 334) and excessive medialward angulation in subcapital amputation of the first metatarsal

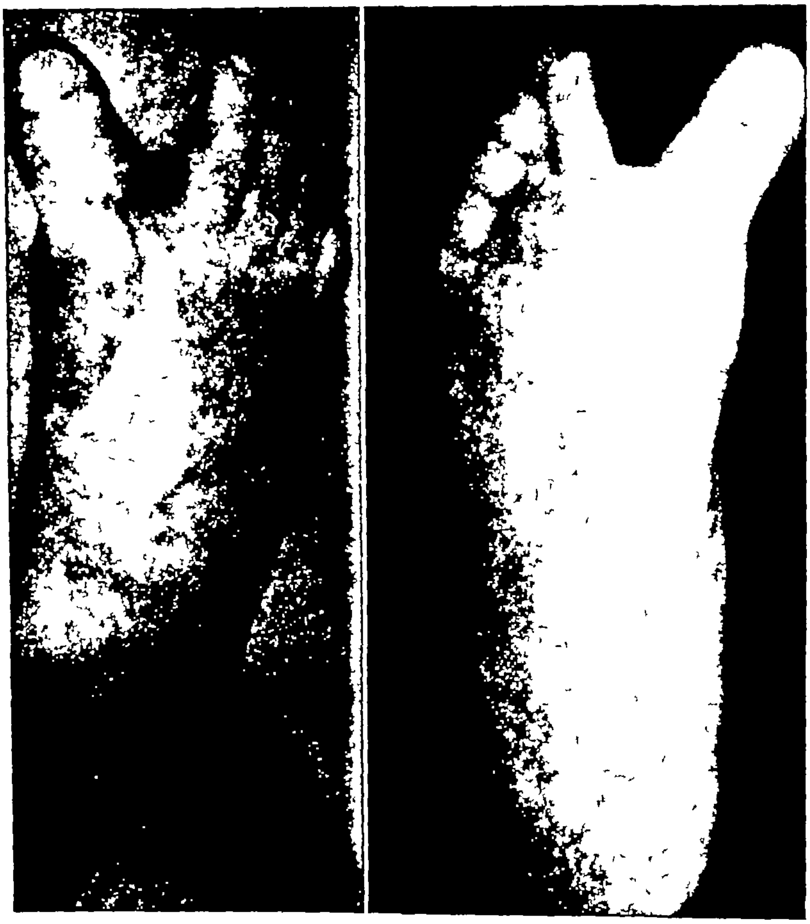


Fig 334 —Postoperative hallux varus due to excessive shortening of abductor hallucis tendon

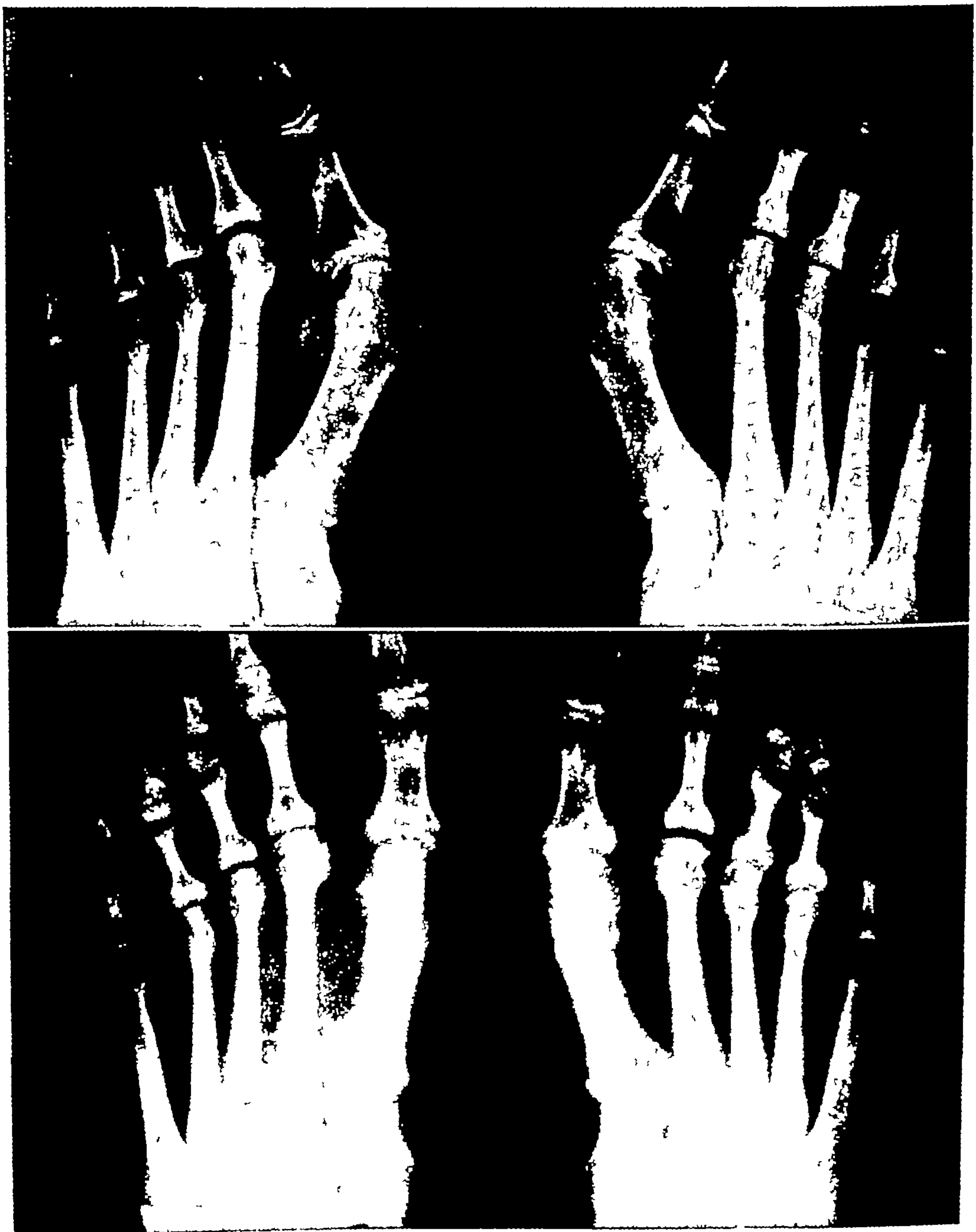
Congenital Types: Treatment.—Congenital hallux varus is rare (Fig 335), only about twenty-eight cases having been reported up to 1957 (Sloane, 1935, Haas, 1938) The case reported by Horwitz (1937) was not a true hallux varus deformity in that the second proximal phalanx was wedged transversely between the head of the first and second metatarsals, forming a cleft foot

Sometimes the disorder is entirely muscular, either a paralyzed or a weak adductor hallucis permits the abductor hallucis to pull the toe into the varus position In another type, the medial articular surface of the great toe joint is so angulated that the base of the proximal phalanx articulates with the metatarsal head at less than a 180 degree angle A comparable condition is sometimes seen in the hallux interphalangeal joint (Fig 336)

ing is ordinarily uneventful. At times a large area of skin around the incision may be affected. This delays healing for a month or longer.

Occasionally a reaction to subdermal sutures (stitch abscess) may not appear until four to eight weeks postoperatively A small bluish fluctuating area appears over the subdermal suture Usually the tissues expel the fluid spontaneously, leaving an aperture through which the suture can be extracted, but occasionally the suture must be removed surgically

A



B

Fig 333 —A, Severe hallux valgus B, Twelve years postoperatively

Congenital types present individual problems In general, when the articular surfaces are normal and the deformity is due to muscular imbalance or fascial contraction, the contracted tissues should be sectioned or lengthened and the opposing side anchored by reinforcing the fascial structures around the first metatarsophalangeal joint to the fascial structures of the second metatarsophalangeal joint If the articular bone is deformed, in addition to altering the soft tissue, the head of the first metatarsal should be reshaped to articulate normally with the proximal phalanx

Postoperative Types: Treatment.—If hallux varus is due to a severe shortening of the tibial capsule and tendon of the abductor hallucis, the capsule and tendon may be lengthened and freed by vertical sectioning Through a second incision over the distal end of the first metatarsal interspace, the capsular structures at the base of the first proximal phalanx are sutured to the capsular structures of the second metatarsophalangeal joint When the deformity is due to an angulation of the head of the first metatarsal, an arthroplasty must be performed and the articular surface of the metatarsal head rounded to form a ball-and-socket joint with the base of the proximal phalanx

Hallux Extensus

Normally, the flexor hallucis brevis is an important opponent of the extensor hallucis longus When this opposing structure is destroyed or severed, the extensor hallucis longus contracts, causing hallux extensus This happens often as the result of sectioning the flexor tendons, especially the flexor hallucis brevis, when both sesamoids have been removed, or when the insertion of the flexor hallucis brevis has been destroyed, or when the base of the proximal phalanx has been amputated The great toe becomes dorsiflexed and its phalangeal joint, hammered

In treatment, whenever possible, attempts should be made to suture the flexor hallucis brevis through a medioplantar incision, however, this is not often possible Lengthening of the extensor hallucis longus will usually permit the deformity to be reduced When the phalangeal joint is hammered, it must be reduced at the same time The technique is discussed under the section on hammertoes, page 347

Hallux Flexus (Dorsal Bunion)

Most cases of hallux flexus are the result of hallux rigidus, except that the proliferation on the dorsum of the head of the first metatarsal is so extensive that it holds the hallux in a constant plantar-flexed position (Fig 337) Only in rare cases, rare because the action of each step normally forces a *dorsiflexion* of the great toe, is the plantar flexion of the great toe held by unusually powerful flexor muscles or paralysis of the extensors (Fig 338)

Lapidus (1940) classified the different types of hallux flexus or dorsal bunion into four groups (1) cases secondary to hallux rigidus, (2) cases due to paralytic deformity of the foot, both flaccid and spastic, (3) cases associated with congenital clubfeet, (4) cases associated with severe congenital talipes plano-

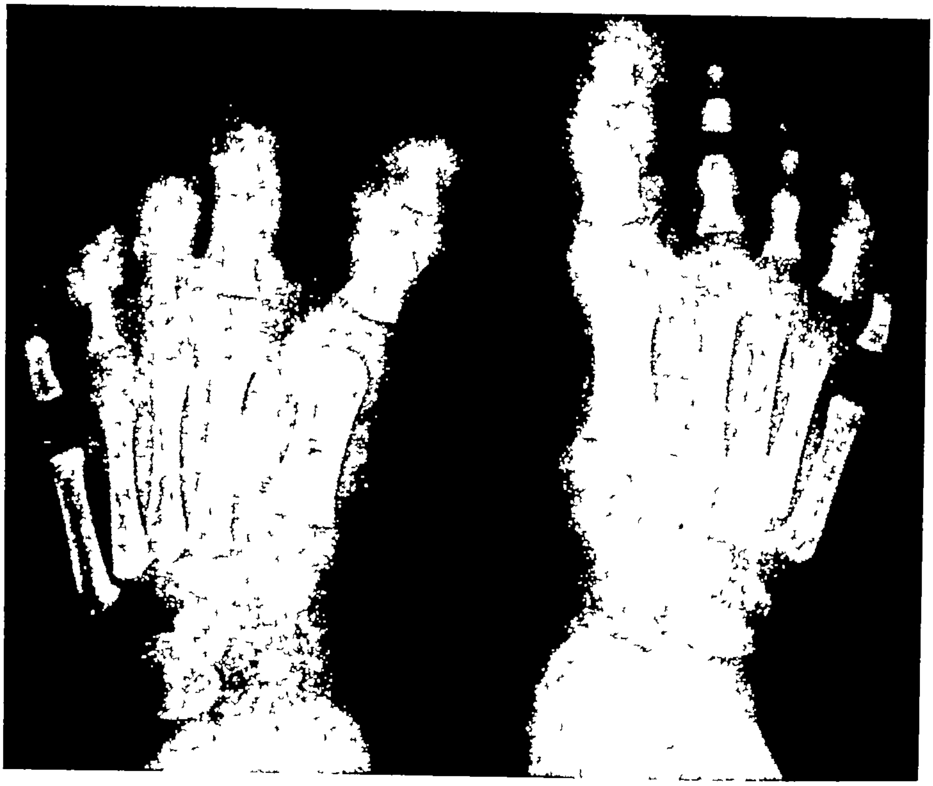


Fig 335—Congenital hallux varus of left foot due to paralysis of adductor hallucis muscle

Fig 336—Hallux varus at interphalangeal joint due to malformation of head of proximal phalanx

A

B.

Fig 338 — *A*, Hallux flexus due to paralysis of extensors from poliomyelitis *B*, Roentgenogram of condition shown in *A* and postoperative result of triple arthrodesis

valgus Lapidus attributed the last group to compensation for a short Achilles tendon.

Surgical approach to treatment should be based on an evaluation of the pathologic-anatomic problem in each case In those cases in which the periarticular structures, including the tendons, are involved, it is sufficient to lengthen tendons that are shortened and shorten those that are lengthened In those cases of bony proliferation (hallux rigidus) limiting the dorsiflexion of the great toe, an arthroplasty of the first metatarsophalangeal joint must be performed

When dorsal bunion is part of congenital talipes planovalgus, Lapidus advised transference of the terminal end of the tibialis anticus tendon into the insertion of the tibialis posticus and the tendon of the flexor hallucis longus into the dorsum of the first metatarsal, just behind its head (Fig 339)

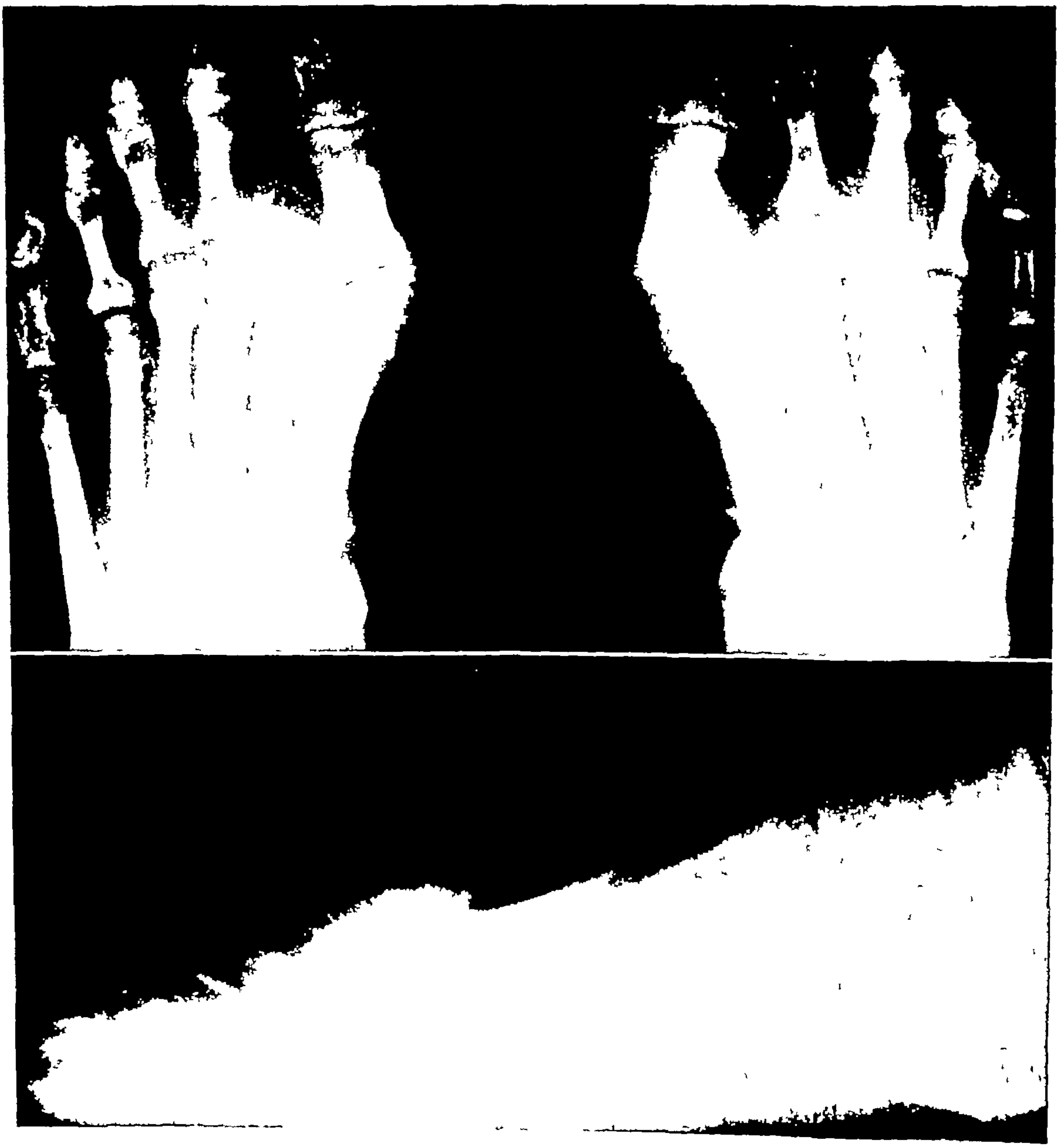
Fig 337 —Hallux flexus resulting from severe hallux rigidus

Hallux Rigidus (Hallux Limitus)

Hallux rigidus, or hallux limitus, is second to hallux valgus in order of prevalence among disabling deformities of the great toe joint Indeed, it may be more disabling than hallux valgus, because in hallux valgus the patient is distressed mainly by the inability to obtain shoes to accommodate the deformity In hallux rigidus the patient cannot obtain relief even when not wearing shoes, because the great toe is locked in a plantar-flexed position Dorsiflexion of the great toe is painful, nevertheless, this movement is essential in every normal step The degree of disability is in direct relation to the degree and extent of the deformity The great toe is either fixed or limited in its normal motion of dorsiflexion, because of a deformity of the articular surface of the head of the first metatarsal in which its dorsal aspect is longer than its plantar aspect, that is, the dorsal margin of the head of the first metatarsal protrudes farther distally than the plantar margin In most cases, the discrepancy is due to an extensive proliferation of bone around the dorsal and lateral sides of the head of the first metatarsal, forming

a collarlike mass (Fig 340), which does not involve the plantar surface of the head of the first metatarsal Often the base of the proximal phalanx is similarly affected In advanced cases, the entire hallux is fixed in a plantar attitude, this forces the patient to walk on the outer border of the foot, thereby inverting the ankle and frequently producing secondary changes in soft tissue and bone of the whole foot

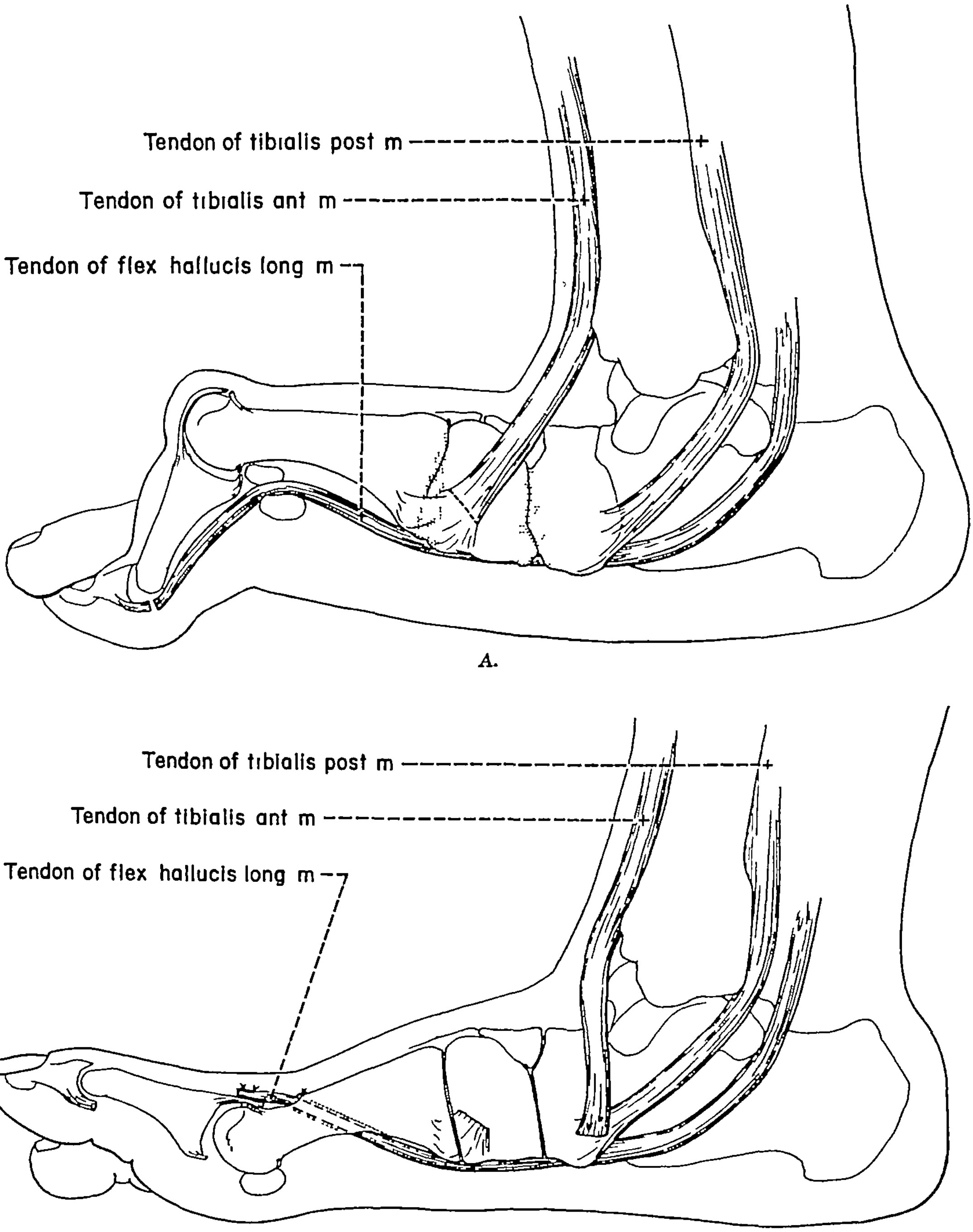
A



B

Fig 340—*A*, Extreme case of hallux rigidus Local traumatic arthritis Violent reaction to persistent injury Third metatarsal and proximal phalanx on right side also reacted to prolonged trauma *B*, Note extensive new growth of bone on dorsum of first metatarsophalangeal joint C, After arthroplasty

(*Fig 340 continued on next page*)



B

Fig 339 —*A*, Schematic drawing of pathologic-anatomic appearance of hallux flexus, according to Lapidus *B*, Lapidus' technique for reduction of hallux flexus Flexor hallucis longus threaded, from plantar to dorsum, through a drill hole in shaft of first metatarsal, sutured on dorsum of bone of first proximal phalanx Terminal end of tibialis anticus tendon sutured into insertion of tibialis posticus

average are influenced adversely by short shoes Short shoes produce hallux valgus in the degree of accentuated ball-and-socket relationship of the joint surface and the amount of fragility or elasticity of the periarticular structures Conversely, the flatter the articular surface of this joint (Fig 312) and the more resistant the periarticular structures are, the more the back pressure of the shoes tends to cause low-grade traumatic arthritis, resulting in proliferative changes of the articular surface, and, eventually, hallux rigidus

Gout or rheumatoid arthritis is a likely cause of hallux rigidus In gout, the great toe is often the only joint of the foot involved, whereas in rheumatoid arthritis, other joints of the foot are also affected Complete arthrodesis of the joint may be consequent to infection after surgical correction for hallux valgus

Treatment.—Treatment for congenital cases differs because the deformity is variable The faulty articular surface of the joint must be carefully evaluated in each case and an arthroplasty performed so that a nearly normal ball-and-socket articular relation may be established between the base of the proximal phalanx and the head of the first metatarsal It is not always feasible to evaluate the problem preoperatively, the true distortion of the joint surface, as reflected in the roentgenogram, can portray only two dimensions, whereas the third dimension of the joint may be largely at fault Unfortunately, it is nearly impossible to obtain a true picture of the mediolateral contour of the metatarsophalangeal joints

False ankylosis results when subcapital amputation of the first metatarsal is performed without rounding the amputated surface

The following arthroplasty procedure corrects acquired hallux rigidus

1 Make a longitudinal incision immediately over the first metatarsophalangeal joint on either side of the extensor hallucis longus tendon, extending it from the middle of the proximal phalanx to a point about the middle of the shaft of the first metatarsal

2 Retract the skin and tendon of the extensor longus hallucis

3 Incise the joint capsule longitudinally and free it from the bone lipping to which it is usually attached, retract the margins

4 Remove all dorsal exostoses

5 Deliver the head of the first metatarsal dorsally Plantar flexion of the great toe at this stage aids delivery

6 Remove excess growth of bone on dorsum and sides of the head The bone is often granitelike in consistency Include some of the normal bone of the head on removal so as to form an accentuated rounded surface but never a pointed surface (Fig 341)

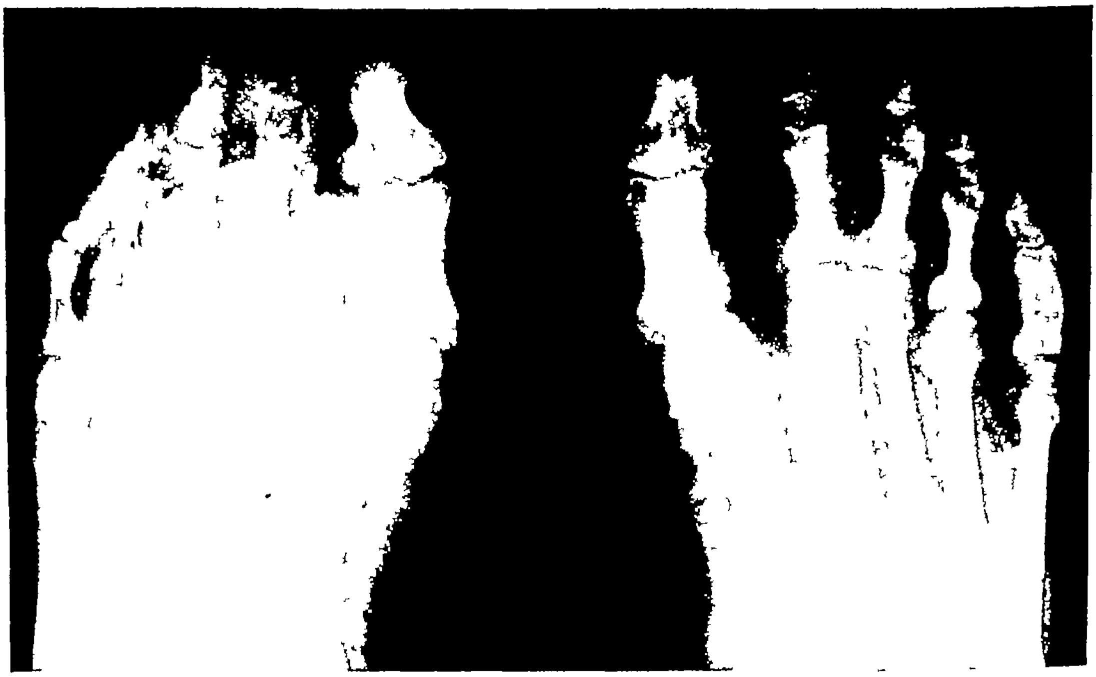
7 Smooth the surface with a rasp If the base of the proximal phalanx is also exostotic, remove excess bone and smooth

8. Instill about 10 mg of hydrocortisone acetate into the joint space to inhibit formation of extensive scar tissue in the capsule

9 Suture the capsule with fine chromic catgut, close the skin as usual Apply a compression bandage while the great toe is overcorrected in a dorsiflexed position

Types.—Miller and Arendt (1940) are of the opinion that hallux rigidus is related to a congenital proximal displacement of the sesamoid Bingold and Collins (1950) studied thirty-three cases roentgenologically and histologically. When they had examined the bone and joint specimens in twelve of their cases, they concluded that there was little to differentiate them into any definite types Mau (1928) believed that an inefficient foot, such as pes valgus or pes valgoplanus, is the forerunner of hallux rigidus.

For convenience, hallux rigidus may be divided into four types· (1) congenital, (2) acquired, as a result of traumatic arthritis; (3) acquired, secondary to one of the general arthritides, (4) acquired, as a postoperative deformity of the great toe joint.



C

Fig 340 (cont'd) —For legend see page 395

Congenital—The congenital type is uncommon It presents an anomalous joint surface, such as an imperfect ball-and-socket relationship between the first metatarsophalangeal articulation Almost always the articular surface of the metatarsal head is flattened, so that each step the child takes is mildly traumatic, producing, over a long period, osseous proliferation

Acquired—The acquired type is commoner It is essentially a traumatic osteoarthritis It is due to prolonged injury to the great toe joint as a result of a combination of any of the following factors (1) a congenitally flattened head of the first metatarsal, (2) unusually strong ligamentous tissues surrounding the joint, (3) occupational extraordinary weight bearing on the great toe joint, (4) repeated use of the great toe as a striking or anchor point The normal great toe joint has a moderate ball-and-socket articulation, and the periarticular structures have normal resistance to external pressure Variations on either side of the

A

B

Fig 342 — *A*, Hallux rigidus, severe dorsal proliferation, joint mouse *B*, Eight months postoperatively

Tailor's Bunion (Bunionette)

The term *tailor's bunion* is applied to any enlargement of the fibular side of the fifth metatarsophalangeal joint The enlargement may be due to one or to a combination of three conditions (1) hypertrophy of the soft tissue overlying the fifth metatarsophalangeal joint, (2) a congenitally wide dumbbell-

A

B

Fig 341 —A, Hallux rigidus B, Same foot, one year after arthroplasty

10 Gentle motion of the great toe joint is instituted on the third or fourth postoperative day and increased in vigor and extent daily for three months to prevent formation of adhesions in the periarticular structures The patient can usually be ambulatory the second or third day

In most cases the patient will be free from pain and have satisfactory motion in the great toe joint in about two months (Fig 342) Sometimes adhesions form and there is residual limitation of motion after complete healing The adhesions are readily broken under light general anesthesia

Hallux rigidus due to one of the general arthritides should be treated conservatively Operation is resorted to only in extreme cases, because recurrence is likely inasmuch as the causative generalized disease remains

Hallux rigidus due to faulty surgical technique may usually be corrected by an arthroplasty of the first metatarsophalangeal joint and severing of the fascial tissues whenever they limit the motion of the joint

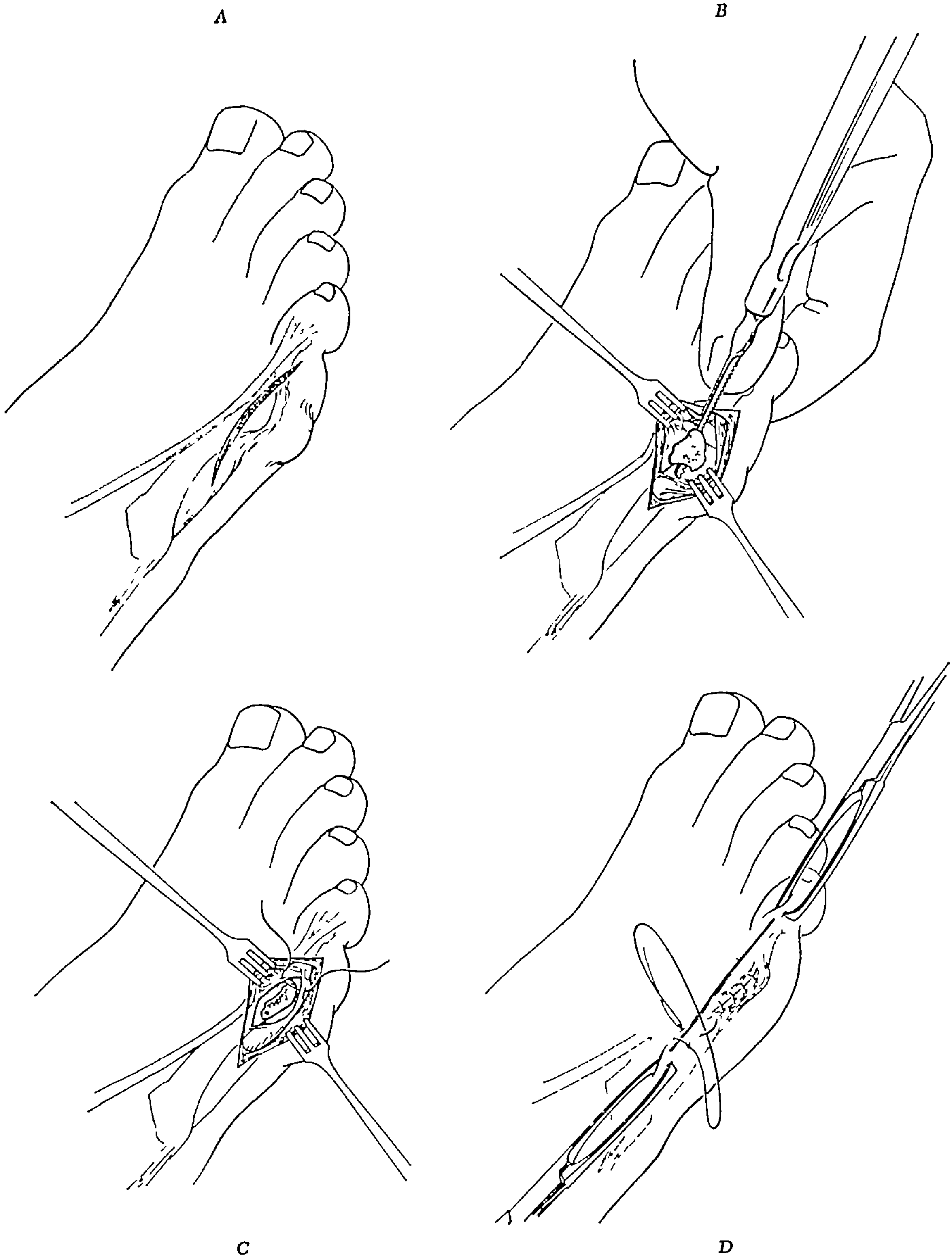
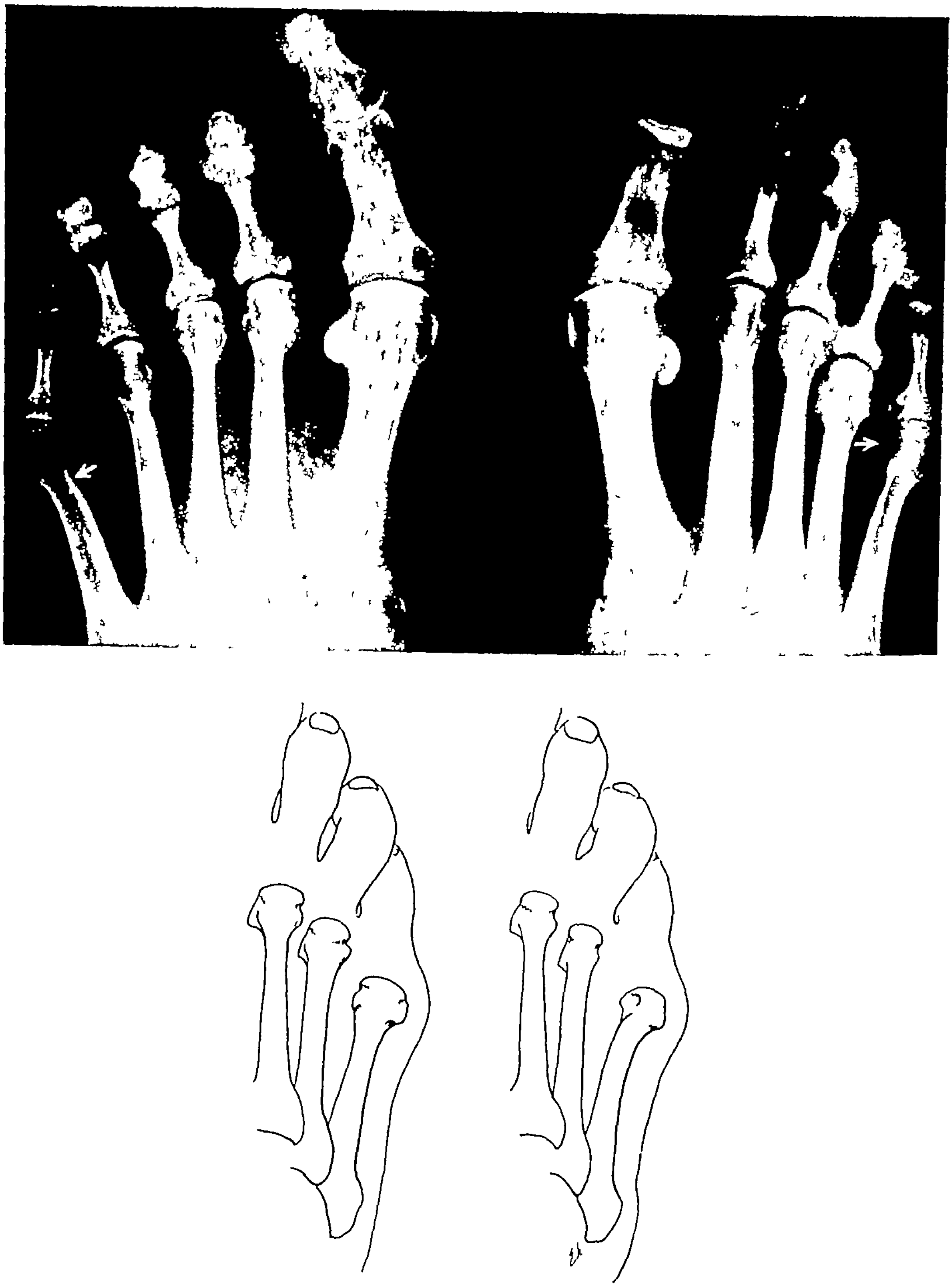


Fig 344 —DuVries' technique for correction of tailor's bunion *A*, Somewhat semielliptical incision over dorsum of fifth metatarsophalangeal joint *B*, Skin, capsule, and tendon of abductor digiti quinti retracted Fibular condyle of fifth metatarsal head amputated with nasal saw *C*, Capsule and tendon of abductor digiti quinti sutured *D*, Skin closed

A

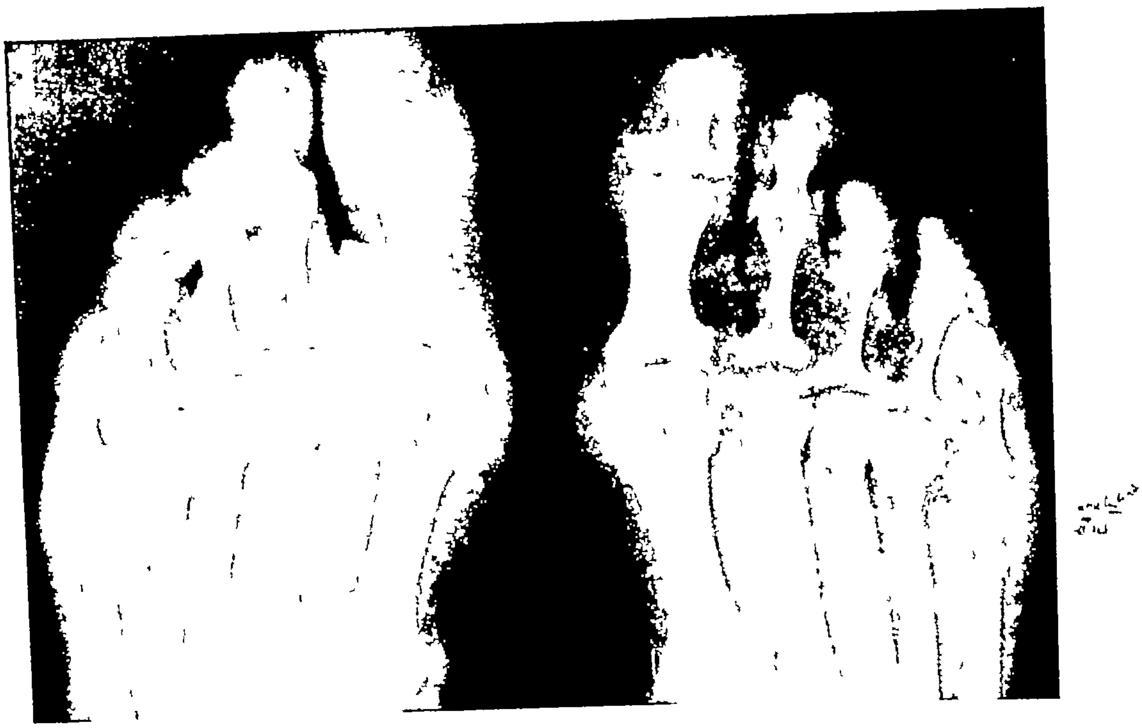


B

Fig 343 —*A*, Tailor's bunion Roentgenogram illustrates two types of bone variation in same person Left, Lateral bending at neck of fifth metatarsal Right, Wide fifth metatarsal head *B*, Schematic drawing of case shown in *A* Left, Wide head Right, Lateral bending of head

metatarsal is normal In such instances the condition results from wearing tight shoes that press against the fibular side of the fifth metatarsal head The congenitally wide metatarsal head is a constantly pressured area even though correctly fitted shoes are worn

Lateral bending of the fifth metatarsal head is the commonest cause of tailor's bunion The fifth metatarsal is normally thin at the neck, it responds readily to pressure, especially during adolescence The wearing of short shoes exerts constant pressure against the fifth toe which in turn is exerted against the head of the fifth metatarsal, thus, the head tends to bend gradually lateralward on its neck



C

Fig 345 (cont'd) —For legend see opposite page

Symptoms —In tailor's bunion the head of the fifth metatarsal is the most prominent point on the side of the foot, consequently, it is subject to the most pressure The area is painful, swollen, and tender and usually is covered by extensive keratosis which may have a deep-seated nucleus under which there may be a degenerative sinus into the joint This area is a common site of chilblains

Treatment —In moderate cases, shielding with pads and carefully fitting shoes alleviate symptoms In protracted cases, operative correction is indicated The *recommended surgical technique* follows

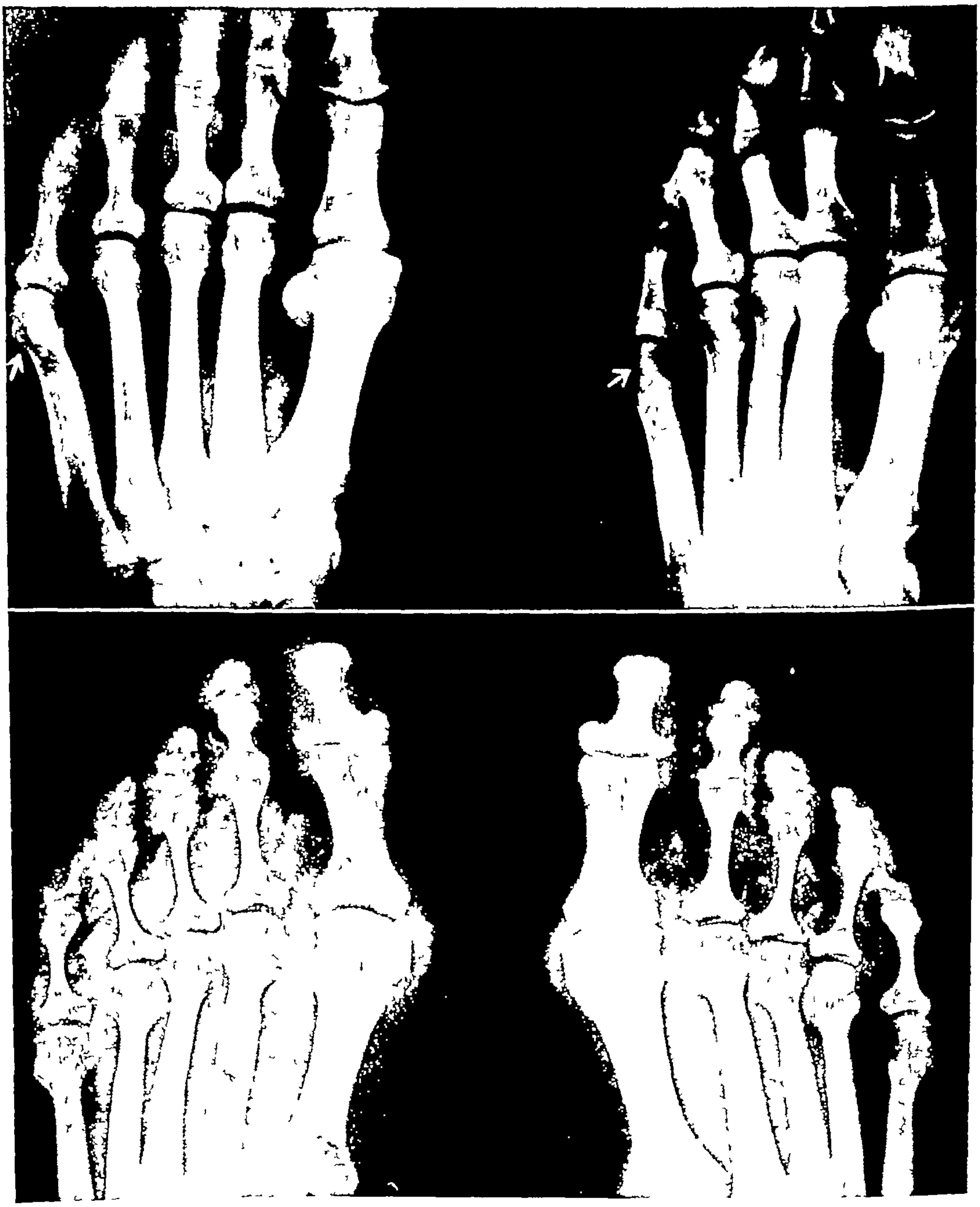
1 Make a longitudinal incision over the dorsolateral border of the fifth metatarsophalangeal joint, extending it proximally from the middle of the proximal phalanx to the juncture of the middle and distal third of the fifth metatarsal (Fig 344, A)

2 Retract the skin and subcutaneous tissue to expose the joint capsule

shaped fifth metatarsal head, (3) a lateral bending of the fifth metatarsal head (Fig 343)

Etiology—Hypertrophy of the soft tissue covering the fifth metatarsophalangeal joint is usually due to either a wide metatarsal head or a lateral bending of this head, however, it is occasionally present even when the head of the fifth

A



B

Fig 345—A, Tailor's bunion Left, Preoperative lateral bending of fifth metatarsal head Right, same foot postoperatively B, Tailor's bunion due to wide fifth metatarsal head C, Same case as shown in B, postoperatively

Fig 346 —Complete static dislocation of second metatarsophalangeal joint and partial dislocation of third metatarsophalangeal joint

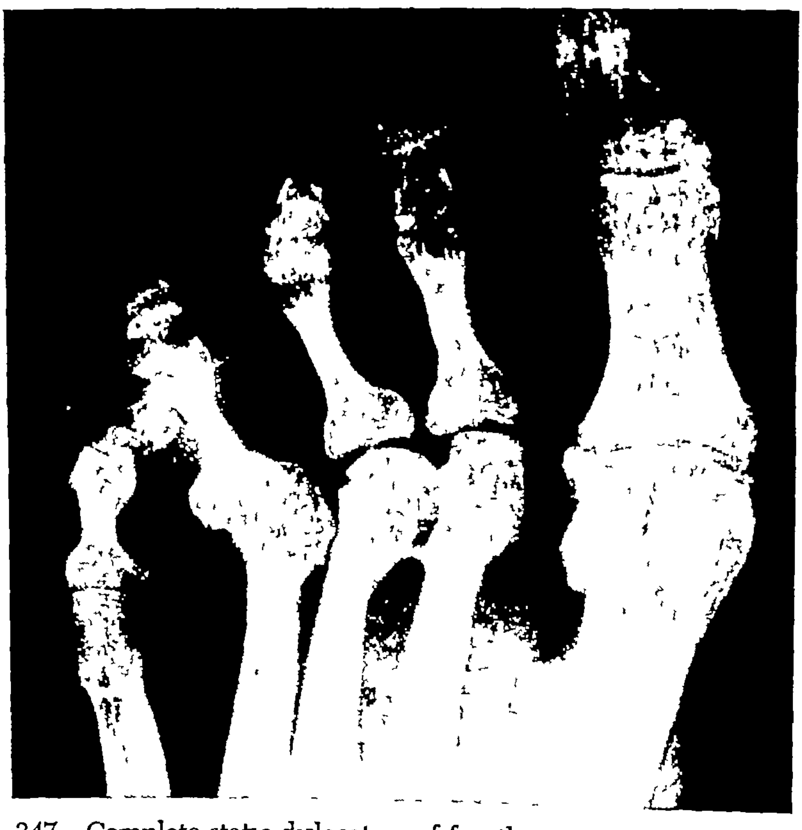


Fig 347 —Complete static dislocation of fourth metatarsophalangeal joint

3 Incise the capsule longitudinally.

4. Denude the fibular border of the capsule, including the tendon of the abductor digiti quinti, from the head of the fifth metatarsal. When retracted with the skin, the fibular condyle of the head will be exposed.

5 Amputate this condylar process longitudinally by means of a nasal saw, beginning at the neck of the bone (Fig. 344, *B*) and round the cut surface with a nasal file. Excessive thickening of the soft tissues overlying the condyle is due to either fibrotic infiltration or adventitious bursae which, when present, should be excised.

6 Close the capsule and skin in layers (Fig 344, *C-D*), making certain to incorporate the tendon of the abductor digiti quinti in the capsule closure, otherwise the fifth metatarsophalangeal joint may become dislocated. Fig 345 shows the end result of the operation

SECOND METATARSOPHALANGEAL DISLOCATIONS

Dorsal dislocation of the second proximal phalanx (Fig 346) is common (Branch, 1937) and is the most frequent static complete dislocation in the forefoot (DuVries, 1956). Single dislocations of the other metatarsophalangeal joints are rare.* Partial or complete dislocation of all the lesser metatarsophalangeal joints is usual in talipes equinus and pes cavus, or clawfoot

Etiology.—The second toe is normally the longest of the five Because the normal position of the toes is in mild dorsiflexion, backward pressure of the short shoe is exerted mostly against the second toe This forces the proximal phalanx to glide over the dorsum of the head of the second metatarsal.

Symptoms.—In most cases of second metatarsophalangeal dislocations, there is an intractable hyperkeratosis (*tyloma*) under the second metatarsal head. This is disabling The base of the proximal phalanx rests directly over the head of the metatarsal, thereby causing the shoe top to accentuate the body weight on the plantar condylar surface of the second metatarsal head On palpation of the second metatarsophalangeal joint, the base of the proximal phalanx can be felt lying on the dorsum of the head of the second metatarsal This deformity is often accompanied by hallux valgus (Fig 348), but it may occur in a foot having a comparatively normal first metatarsophalangeal joint

Treatment.—Open reduction of the dislocation corrects most cases and improves the remainder The *recommended surgical technique* follows

1. Make a longitudinal incision immediately over the dislocation.

2 Retract the skin and extensor tendon In some cases, this tendon has to be severed because of severe contraction

3 Open the capsule longitudinally Section all adhesions.

4 Reduce dislocation and hold in place

5 Suture the dorsal capsule and fascia remnants with fine chromic gut while the toe is held in reduction until the reduction maintains itself

---

*I have seen one case in which the fourth metatarsophalangeal joint (Fig 347) was completely dislocated but none of the fifth, whereas my records show 100 cases of static dislocation of the second metatarsophalangeal joint —H L DuVries

6 Suture the skin

7 Place a gauze plug over the reduced joint, on top of which place part of a tongue depressor, apply a compression bandage This will maintain the toe in reduction during the healing process

8 Replace splinting after each dressing for about five weeks

The linear scar in the capsule and fascia, which will form over the dorsum of the joint, will prevent further dislocation (Figs 332 and 349) In a few cases, the proximal phalanx cannot be held in a reduced position because the head of the metatarsal has undergone angular atrophy on its dorsal aspect This atrophy can be seen only during the surgical procedure In such instances, the head of the metatarsal should be rounded, so that it will articulate normally with the base of the proximal phalanx

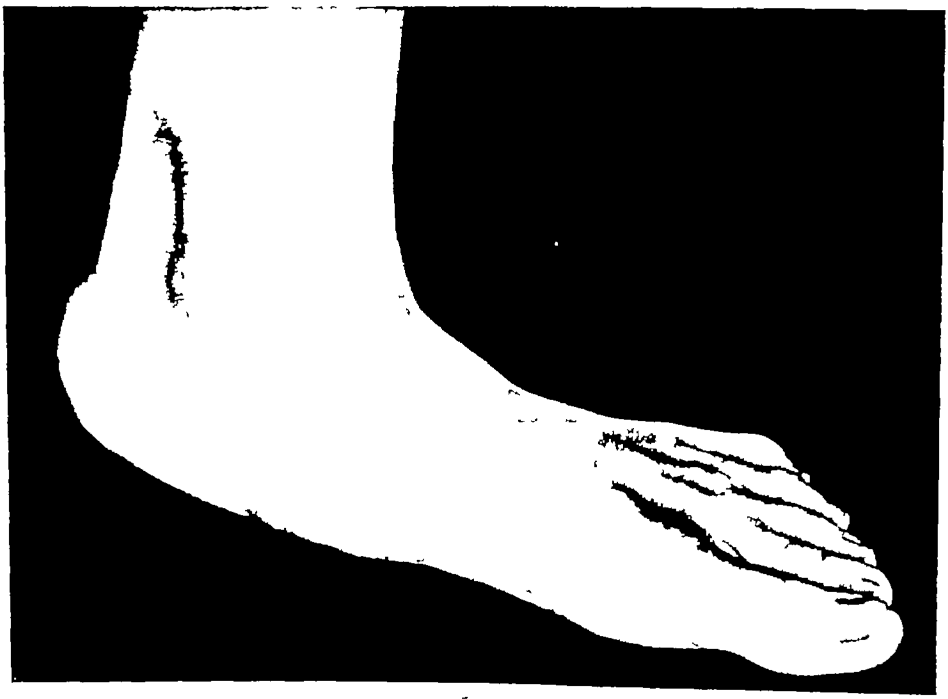


Fig 350 —Flatfoot and eversion and abduction of forefoot

Flatfoot (Pes Planus)

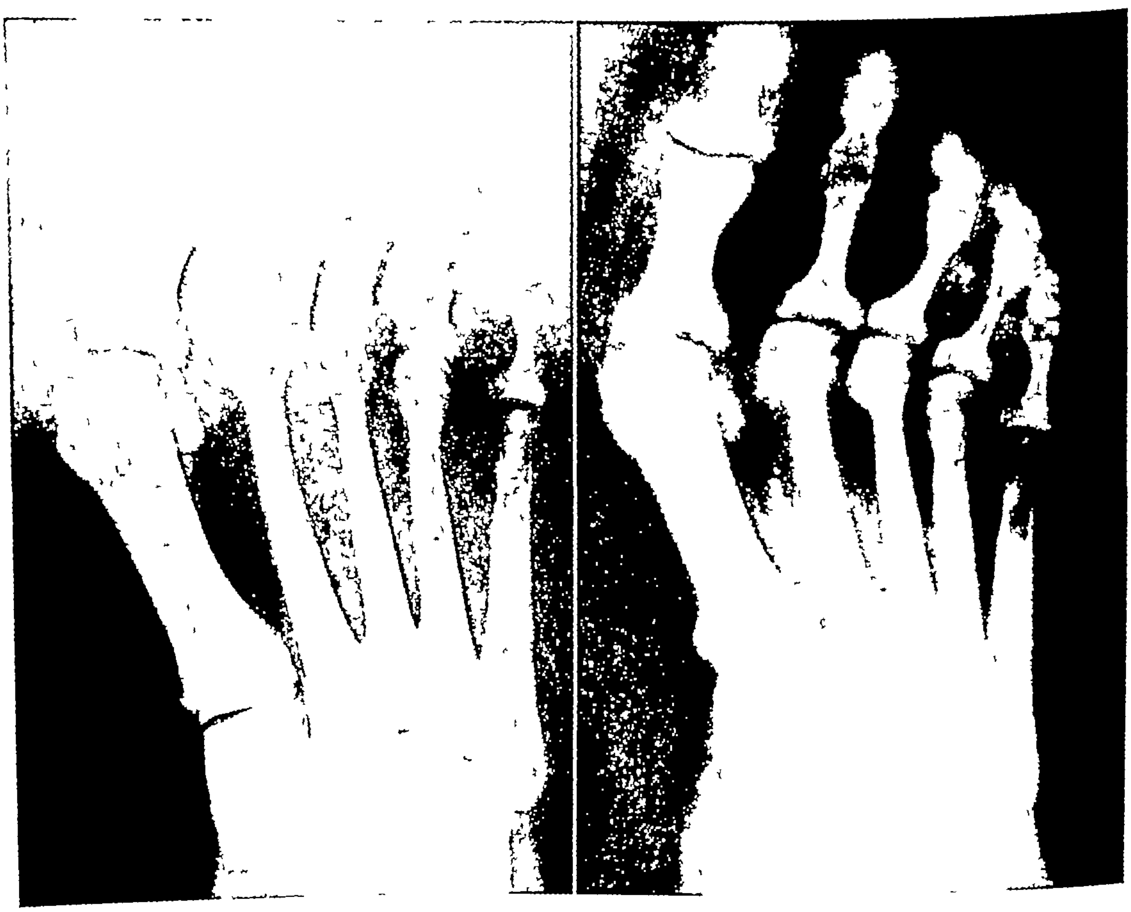
Flatfoot, weak foot, or fallen arches, as the disorder is variously called, is difficult to classify There is no known standard by which the longitudinal arch may be considered flat, normal, or high Some primitive peoples in Africa and Australia are all flatfooted When they have painful feet, however, it is only as a result of injuries, whereas in our modern society, many people with so-called normal arches have painful longitudinal arches on weight bearing

Harris and Beath (1948) attributed the weak flatfoot to deficiencies in structure of the talus and calcaneus, conversely, strong and well-shaped feet are attributed to the result of tarsal bones so shaped and so integrated into one another that they cannot shift when weight is imposed on them

The symptomatic weak foot, then, may be flat or may have a high longitudinal arch, especially at rest The flatfoot usually has a degree of abduction

Fig 348—Complete static dislocation of second and third metatarsophalangeal joints, and hallux valgus

A



B

Fig 349—*A*, Complete static dislocation of second metatarsophalangeal joint *B*, Two years postoperatively

The flaccid type is undoubtedly due to excessive stress on the foot as a result of overweight and of poorly fitting shoes during adolescence As Nature attempts to force the foot at rest, as happens in fractures or chronic joint disease, a compensatory reflex spasm of the peroneal muscles takes place

The rigid type is often the end result of long duration in cases of the flexible type, sometimes it is due to congenital tarsal anomaly (page 429) Harris and Beath thought that the rigid type is always due to an anomalous talocalcaneal bridge or to a calcaneonavicular bar Webster and Roberts (1951) supported this theory Either or both anomalies may be present

Kidner (1929) believed that a number of these spastic flatfoot cases are related to the presence of an accessory navicular or prehallux Because the posterior tibial tendon is usually attached to the prehallux, its normal support of the longitudinal arch is lost It is true, as Kidner says, that a prehallux is sometimes the cause, not because the support of tibialis posticus is lost, but rather because of irritation from the movement of the prehallux against the body of the navicular That is what produces a reflex peroneal spasm in an attempt to avoid irritation of the head of the talus (See Fig 288 and Fracture of Tuberosity of Navicular, page 332) Lapidus (1946) regarded spastic flatfoot as being due to a lesion of the interosseous talocalcaneal ligament in the subtalar joint

The peroneus longus and brevis are the muscles that are in spasm, but there is evidence that the peroneus brevis is more productive of deformity, as pointed out by Blockey (1955), who concluded, after studying thirty cases of spastic flatfoot, that tarsal coalition probably contributed to but did not necessarily produce a rigid flatfoot inasmuch as the anomalies are often absent in rigid flatfoot Merryweather (1955), in his study of sixty cases of spastic flatfoot, observed tarsal anomalies in only 40 per cent, fifty-two out of the sixty were treated successfully by conservative measures, such as immobilization with overcorrection "suited equally to those with or without anomalies "

Many short, squatty, overweight adolescents have endocrine deficiency, bordering on cretinism Under glandular therapy, coupled with proper footgear and shoe wedging, many respond * There is a relationship between this malady and tarsal coalition (See page 429)

Treatment of Types of Flatfoot.—The type of flatfoot that is due entirely to muscle spasm, after anomalies such as bars or bridges or prehallux have been ruled out, can usually be controlled by conservative measures To differentiate between the flexible and rigid types, it is often necessary to place the patient under general anesthesia for complete voluntary muscle relaxation If the deformity is readily reduced under anesthesia, a plaster cast with walking caliper may be applied with the foot overcorrected Many will remain corrected and require only corrective shoes for a time after removal of the cast Some will require repetition of overcorrection under anesthesia and immobilization

In a few cases, injection of 10 to 20 ml of 1 per cent procaine hydrochloride relieved the peroneal spasm †

*Personal observations
†Author's cases

of the forefoot (Fig 350) and eversion of the ankle The tendo achillis may be shortened and pulled at an angle instead of following a plumb line (Figs 42 and 43) Pain is usually chronic but may be acute The long, narrow, flaccid type of foot with so-called normal arch is likely to become symptomatic

Only the flatfoot that is symptomatic needs medical attention (Fig 351) A variety of alleviating devices is available corrective shoes, wedging of shoe or inlays of leather, metal or plastic materials, or both wedging and inlays, as recommended by Dively (1934), Graham (1939), Bingham (1944), Ilfeld (1944), Lewin (1947), and Hauser (1950). In acute cases, adhesive strapping to hold the foot in adduction and eversion usually gives immediate relief

A few cases may become intractable Appliances do not give relief, stabilization is the only resort.

Flatfoot in children is discussed in Chapter 17, Anomalies and Congenital Defects

Fig 351 —Symptomatic severe flatfoot (rockerfoot), which required major stabilization

Peroneal Spastic Flatfoot (Spastic Pes Planus)

Spastic flatfoot has posed many questions since Jones (1916) called attention to the prevalence and disabling effect of the disorder The term is loosely applied to a group of conditions arising from different causes It usually begins during adolescence and is characterized by severe pes planus, an abduction of the forefoot, and an eversion of the ankle, caused by constant spasm of the peroneal muscles

Two distinct types of the condition are recognized the rigid and the flexible Under anesthesia, the spasticity of the flexible type disappears and the foot can be placed in a normal position, whereas the rigid type remains fixed Harris and Beath (1948) reported that among 3,600 candidates for Army enlistment, 74, or 2 per cent, of the rigid type were observed and 217, or 6 per cent, of the flexible type

Etiology.—The cause of spastic flatfoot is not entirely understood, probably because the condition is a symptom complex rather than a disease entity Many factors are predisposing, they may be present singly or in combination But the same predisposing factors may be present without producing the syndrome

L'Episcopo and Sabatelle (1939) reported that they observed sixteen patients with flatfoot after operation by Hoke's procedure, 68 per cent had obtained good results and 32 per cent fair results Butte (1937) reported on seventy-six feet operated on by Hoke's procedure, results were satisfactory in about 50 per cent Jack (1953) performed Hoke's operation for flatfoot in forty-six cases and obtained 82 per cent satisfactory results.

Zadek (1935) recommended a wedge osteotomy of the talocalcaneal joint. White (1940) removed a wedge of bone from the medial side of the neck of the talus and then inserted it into an opened osteotomy on the lateral side of the neck of the calcaneus Harris and Beath (1948) advised a subtalar and talonavicular fusion

No one procedure can be used in all cases Conservative measures should always be exhaused before resorting to surgical procedure of osseous disorders. Each case should be evaluated and a procedure selected that is best suited to the case

Generally, Schoolfield's shortening and reinforcement of the deltoid ligament and Hoke's navicular-cuneiform bone graft are the most useful operations for the flexible type of flatfoot, whereas triple arthrodesis must usually be resorted to for the rigid type

Talipes

Major foot and ankle deformities are either congenital or acquired Until the early part of this century, a large number were congenital "The time to treat a congenital clubfoot is immediately after tying the natal cord" has become a maxim in modern medical training Reports on the varieties of talipes are extensive and often controversial It is the one topic regarding surgery of the foot which has received thorough attention in publications

Clawfoot

Clawfoot, also known as *hollow foot, pes cavus,* or *pes arcuatus,* is a distinct deformity (Fig 353) which differs from congenital talipes equinus or calcaneus as well as from paralytic talipes The typical condition presents an exaggeration in the height of the longitudinal arch and slight shortening of the foot, prominent metatarsal heads, clawing of the toes, loss of flexibility in all the joints of the foot, reduction of treading surface, and often a limited dorsal flexion at the ankle joint The deformity is largely one of the forefoot, with typical dorsal contracture of all the toes and dislocation of the metatarsophalangeal joints The apex of the deformity on the inner border of the foot is usually the navicular cuneiform joint. There are many grades of the cavus, ranging from a simple high arch and dorsally deformed midfoot area to an extreme concavity of the longitudinal arch Often the forefoot is dropped, and the deformity may be accompanied by a true equinus attitude brought about by contraction of the calf muscles, it may also be combined with varying degrees of malposition of the calcaneus as a result of lengthening of the heel cord

The foot is commonly inverted in a clawfoot appearance so that too much weight is thrown on the outer border, often causing the base of the fifth metatarsal

Recurrent cases of the flexible type of flatfoot without peroneal spasm may be corrected by Schoolfield's (1952) operation (Fig. 79), which shortens and reinforces the deltoid ligament.

In selected cases in which the tibialis anticus has become paralyzed or has lost most of its power, the peroneus longus may be transferred into the insertion of the tibialis anticus or into the body of the medial cuneiform Sectioning the peroneus brevis will further reduce the pull of the foot in abduction

Treatment of the rigid type is difficult Numerous stabilization procedures and wedge osteotomies have been reported. After a review of the end results of several operative procedures on 102 feet of children for the correction of flatfoot, Crego and Ford (1952) were forced to conclude that arthrodesing operation for flatfoot should never be done only for cosmetic reasons and is only justified to alleviate pain Weil (1955) likewise reviewed the end result of treatment in 400 cases (561 feet) of rigid flatfoot, 189 had been treated conservatively and the remainder by a variety of surgical procedures, such as tendon transference and osteotomies. In only 36 per cent were results of operation satisfactory

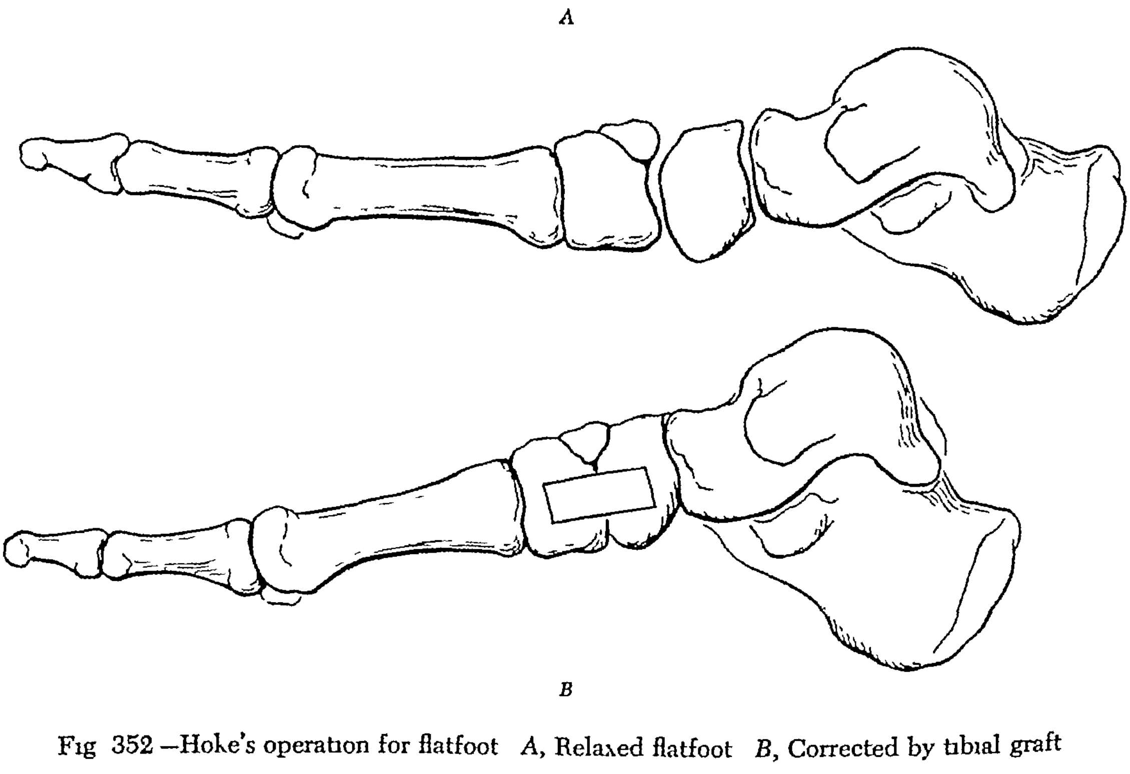


Fig 352 —Hoke's operation for flatfoot *A*, Relaxed flatfoot *B*, Corrected by tibial graft

Hoke (1921, 1931), an early student of foot deformities, proposed the following stabilizing procedure for flatfoot (1) lengthen the tendo achillis, (2) through an incision along the medial border of the foot, exposing the navicular-cuneiform joint, denude and reflect the ligamentous covering, (3) force and maintain the foot in extreme plantar flexion while excising a rectangular block of bone from the navicular and medial cuneiform joint on the longitudinal plane, (4) remove a segment of cortical bone of equal dimension from the tibia and fit it into the rectangular slot bridging the joint (Fig 352)

The aim in treatment for each patient should be to relieve symptoms, correct the deformity, and prevent recurrence. The trend has been toward radical procedures, even in mild cases Every foot, however, should be treated as an individual problem. Inasmuch as a method applicable to all cases cannot be offered, an outline of treatment based on the degree of deformity is herewith suggested

Slight Deformity—In mild and especially early cases of flexible feet with moderate cavus which disappears on weight bearing, the permanent contracture of the plantar structures can be prevented The cavus and clawing of the toes can be kept to a minimum by careful fitting of proper shoes having a low heel and, when indicated, a metatarsal bar and wedging Exercises are recommended to increase flexibility of the foot and stretching of the toes and muscles of the calf

Moderate to Severe Deformity—Operative correction has been suggested variously by tendon relocating and lengthening, fasciotomy, plantar muscle denervation, anterior tarsal wedge osteotomy, and even subtalar arthrodesis Hibbs (1919) suggested transferring of the extensor hallucis longus and extensor digitorum longus into the cuneiforms Steindler (1917) fostered stripping of the plantar fascia from the plantar surface of the calcaneus to dispose of its bowstring action on the longitudinal arch In moderate cases, Cole (1940) employed Steindler's fasciotomy, followed by the use of a Thomas wrench and Hibbs' relocation For extreme deformity, he recommended an anterior tarsal wedge osteotomy Alvik (1954) extended the wedge osteotomy forward to include a wedge resection of the first and second cuneiforms and base of the first and second metatarsals

Evaluation—It is questionable whether soft tissue operations alone, such as Steindler's fasciotomy or tendon transference, or both combined, ever corrects any of the clawfoot deformities Steindler himself recommended the procedure only in "the mildest of cases" Brockway (1940), in discussing Steindler's operation, stated, " it is surprising how little correction can be obtained, and frequently relapse occurs ."*

No single procedure is suitable for all cases of clawfoot.

Metatarsus Adductus (Metatarsus Varus)

Metatarsus adductus (Speed and Knight, 1956) is a common deformity which differs from other congenital varus deformities in that the tarsus is normal and there is no tibial torsion The deformity is essentially in the tarsometatarsal articulation (Fig 354) The forefoot is held in adduction and inversion, the longitudinal arch is higher than normal, and weight is borne by the base of the fifth metatarsal The deformity is commoner than is realized and appears to be increasing in frequency McCormick and Blount (1949), who call the deformity "skewfoot," and Kite (1950), who reported 300 cases, called attention to the increasing incidence

*Brockway, A Surgical Correction of Talipes Cavus Deformities, J Bone & Joint Surg 22 81-91, Jan, 1940

to become a weight-bearing, pressured point In clawfoot, the base of the first three metatarsals is so high that the heads of the metatarsals strike the ground pointedly, consequently, most patients have heavy callosities and sometimes traumatic ulcers under one or more of the heads

Etiology.—The cause of clawfoot is still controversial, although the deformity is common The theories of etiology advanced most are congenital origin, acute illnesses of childhood, constriction of the feet during growth by improper shoes, intrinsic and extrinsic muscular imbalance, paralysis in the foot consequent to poliomyelitis, diseases of the central nervous system, heredity, trauma, and infection

Fig 353 —Clawfoot Note high longitudinal arch and contraction of all toes

Clawfoot appears oftener during early childhood or adolescence and about as frequently in boys as in girls No relationship to race or social status has been demonstrated It may be associated with many known disorders or may occur independently Saunders (1935) concluded that congenital abnormalities of the spinal cord often produce clawfoot but that cases of truly independent congenital origin, in the sense that it is seen at birth or during infancy, as are the congenital talipes, are rare, and general experience proves this Kochs (1927) reported a case in which laminectomy was performed for spina bifida, spontaneous correction of foot deformity ensued (See page 287)

Symptoms.—The degree of deformity is variable Symptoms of the condition are inability to walk or stand for long periods, easily tiring feet, severe excrescences, which may ulcerate, on the ball of the foot and on any or all of the toes, and often, heavy callosity under the base of the fifth metatarsal due to foot inversion

Treatment.—Garceau and Brahms (1956) reported forty-seven cases of pes cavus which they treated by denervation of the plantar muscles In one case of complete paralysis of all the leg muscles, they observed a progressive cavo-adductus deformity and decided to denervate the plantar muscles This arrested the progress of the cavus deformity

crest of the tibia at the juncture of its middle and lower thirds. Pull the tendon out through this incision.

3 Make a third horizontal incision immediately over the cuboid

4 At this point make a tunnel with a long firm probe or with straight uterine forceps, connecting the second with the third incision Be certain that the tunnel underscores the transverse crural and cruciate crural ligaments.

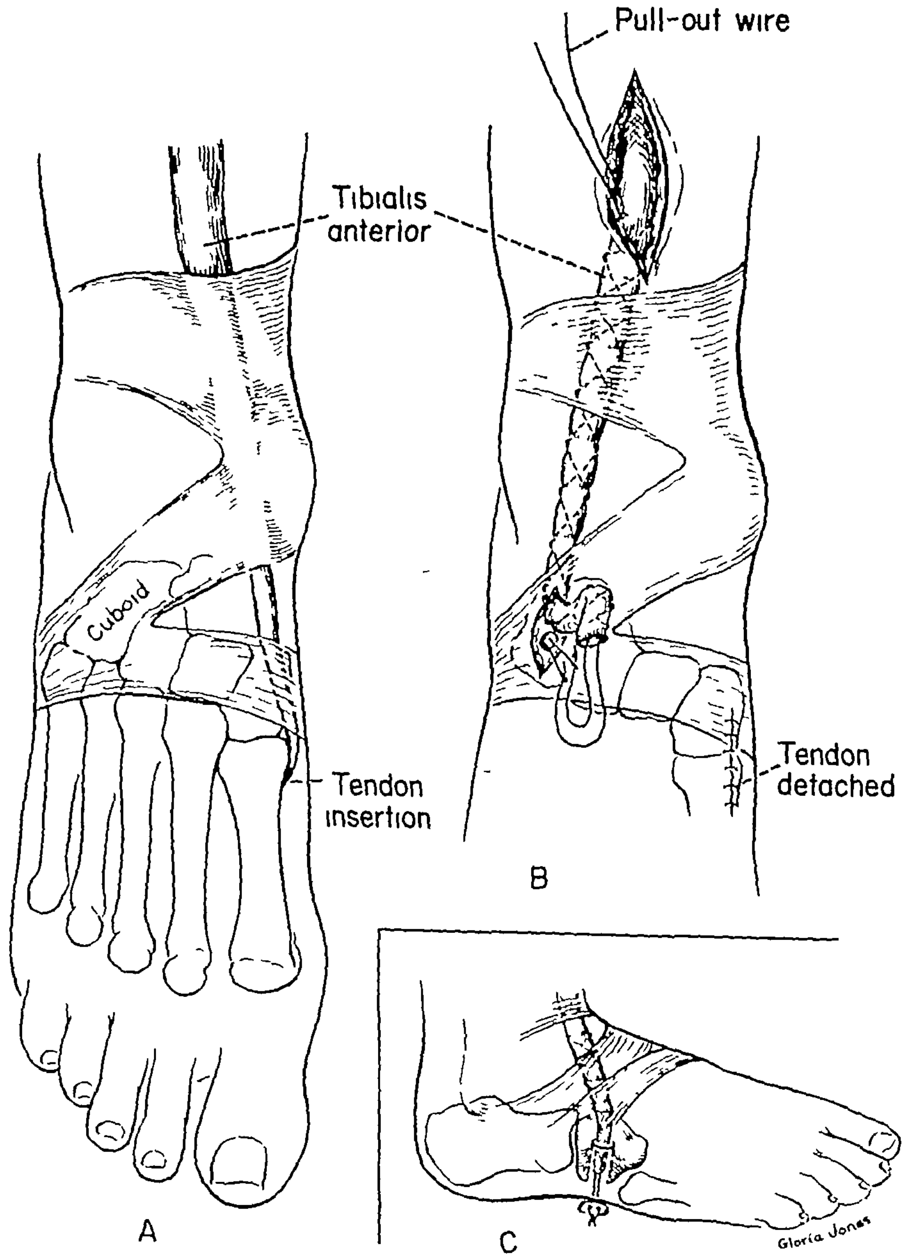


Fig 355 –Transference of tibialis anticus into cuboid

5 Thread a stainless steel wire through the entire length of the exposed tibialis anterior tendon and insert a pull-out wire through the most proximal loop of the threaded wire

6 Attach the terminal ends of the threaded wires to a Bunnell tendon guide, thread the guide through the tunnel to the cuboid

Etiology.—The deformity is usually congenital but is not always observed at birth In some cases recognition of the deformity comes when the child begins to walk, in others, not until the age of 3 or 4 years Often treatment is not sought until adulthood.

The tibialis anticus is overactive in metatarsus adductus, an abnormal insertion of this muscle is probably often a basic cause Normally this muscle is inserted equally into the base of the first metatarsal and first cuneiform Sometimes the insertion is primarily into the first metatarsal and may even give off tendinous slips to the head of the first metatarsal In such a state it does not have an opponent muscle to keep the forefoot in alignment, thus, the forefoot goes into varus

A *B*

Fig 354 —*A*, Metatarsus adductus in an adult *B*, Same foot after stabilizing first metatarsal-first cuneiform joint

Treatment —In infancy and early childhood the deformity responds, in most cases, to abductor shoes or plaster-cast correction During adolescence, transference of the tibialis anticus into the cuboid corrects most cases By the time of adulthood, the deformity has become static, requiring osteotomy and arthrodesis of the first metatarsal and first cuneiform articulation and correction of an abnormal insertion of tibialis anticus

Technique for Transference of Tibialis Anterior Into Cuboid (Fig 355) —

1 Make a horizontal incision over the medioplantar aspect of the first cuneiform Detach the tibialis anterior tendon from its insertion

2. Make a second linear incision about 4 cm long on the lateral side of the

Technique for Arthrodesis of First Metatarsal-First Cuneiform Joint—

1 Make a semielliptical incision over the medial side of the first metatarsal-medial cuneiform joint The crest should be dorsally

2 Dissect the skin margins and retract

3 Detach the tibialis anterior tendon from its insertion and free for about 6 cm proximally Lay it aside.

4 Incise the ligaments and capsule horizontally over the base of the first metatarsal and first cuneiform and retract the margins

5 Make a wedge osteotomy with its base fibularward at the first metatarsal-first cuneiform articulation Make the wedge so that the two bones will fuse in a straight line A pin, screw, or staple may be used to maintain the bones in coaptation (Fig 354)

6 Suture the ligaments and fascia Suture the free end of the tibialis anterior to the covering of the medial side of the first metatarsal-first cuneiform articulation

7 Close the skin While holding the foot in abduction and eversion, apply a plaster mold to the foot and leg Bivalve the mold in twenty-four hours

8 Remove the skin sutures in ten days, apply a walking plaster boot with the foot held in mild abduction and eversion, for six weeks

9 Maintain correction by a sturdy walking Oxford, which the patient wears for six months

Drop Foot

Symptoms.—Paralytic drop foot is a condition wherein the foot is held mostly in an equinus but in some cases in a varus attitude (Fig 357) It differs from paralytic talipes equinus in that in typical paralytic drop foot, the foot, although limp, can be dorsiflexed passively, because the calf muscles are not contracted, nor are the ligaments or bones fixed in this deformed state Sensory function is usually normal but may be diminished The patient is unable to dorsiflex the foot, trips easily, and stumbles frequently unless the foot is kept cleared of the ground

Etiology.—The condition is usually due to paralysis of the anterior tibial and peroneal muscles, often as a result of poliomyelitis and sometimes as a result of injury to the common peroneal nerve, the common peroneal nerve being superficial as it courses around the neck of the fibula Injury to the neck of the fibula, hence to the peroneal nerve, may be iatrogenic or the result of violence, or impairment may be due to disease or injury to the lumbosacral roots One case was proved conclusively to have been functional *

Treatment.—In cases in which impairment of the common peroneal nerve is reversible, treatment should be undertaken immediately In static cases, a drop foot brace may be recommended if surgical correction is contraindicated Many designs for such braces are available, they depend essentially on a metal spring to force the foot into dorsiflexion

Operative intervention may require tendon transference or Campbell's (Speed and Knight, 1956) or Gill's (1933) posterior bone block procedure (Fig 358) †

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*Personal observation

†My own experience with posterior bone blocks has been satisfactory—H L DuVries

7 Close the first and second incisions, leave the pull-out wire on the skin surface of the second incision

8 Drill a 7 mm dorsiplantar hole through the posterior aspect of the cuboid, so as to clear the peroneus longus tendon Save the bone dust produced by drilling

9 Attach the terminal ends of the threaded wires to a Keith needle and guide through the drill hole and through the plantar surface

10 Remove the threaded wires from the Keith needle and pass through the holes of a button. Tie while the foot is held in eversion

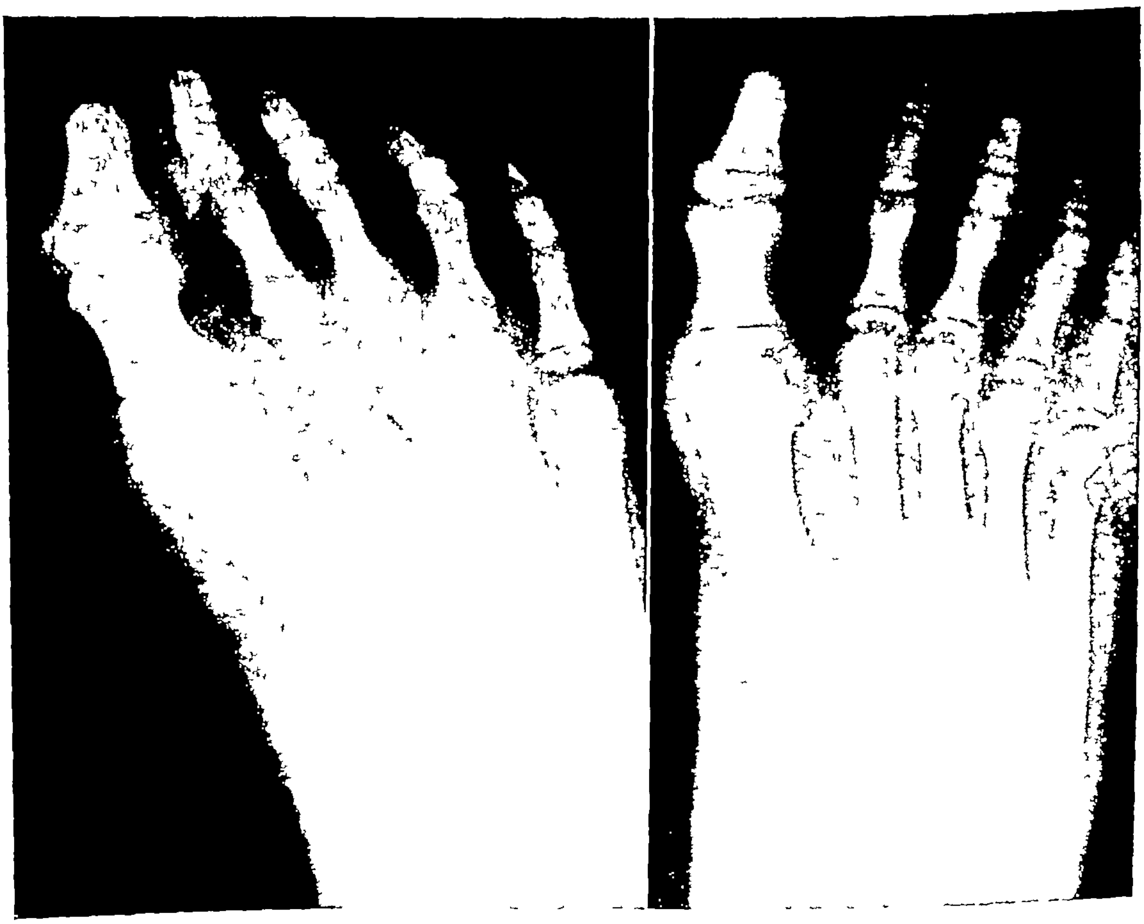
11 Pack the bone dust from the drilled hole around the dorsal entrance of the tendon and close the skin

12 Immobilize the foot and leg in plaster Bivalve the plaster mold after twenty-four hours

13 In ten days, remove the skin sutures, apply a walking plaster boot

14 During the eighth postoperative week, remove the cast By cutting the wires over the button, it is possible to remove all the wires in the tendon by the pull-out wire

The foot gradually assumes a normal attitude (Fig 356)



A *B*

Fig 356 —*A*, Metatarsus adductus in boy, aged 12 years *B*, Nine months after transferring tibialis anticus into cuboid

Jones, Robert Notes on Military Orthopedics, London, 1917, Cassell & Co , Ltd
Lapidus, P W Operation for Correction of Hammer-Toe, J Bone & Joint Surg 21 4, Oct , 1939
Lorenz, H Operative Therapy of Hallux Valgus, Hammer-Toe, and Unbearable Corns, Wien med Wchnschr 79 713, May 25, 1929
Michele, A A , and Krueger, F. J Correction of Hammer Toe, Mil Surgeon 103 52-53, 1948
Selig, S Hammer-Toe A New Procedure for Its Correction, Surg Gynec & Obst 72 101-105, Jan , 1941
Taylor, R G An Operative Procedure for Treatment of Hammer-Toe and Claw-Toe, J Bone & Joint Surg 22 3, July, 1940
Tierny, A Hammer-Toe The Cup and Ball Procedure, J. Bone & Joint Surg 20 4, Oct , 1938
Wagner, L C Operative Correction of Extreme Flexion Contraction of the Great Toe, J Bone & Joint Surg 16 914-918, Oct , 1934
Young, C S An Operation for Correction of Hammer-Toe and Claw-Toe, J Bone & Joint Surg 20 3, July, 1938

Overlapping Toes

Lantzounis, L A Congenital Subluxation of Fifth Toe and Its Correction by Periosteocapsuloplasty and Tendon Transplantation, J Bone & Joint Surg 22 1, Jan , 1940
Lapidus, P W Transplantation of Extensor Tendon for Correction of Overlapping Fifth Toe, J Bone & Joint Surg 24 3, July, 1952
Stamm, T T Elevated Fifth Toe, in Chapter on Surgery of the Foot, Brit Surg Practice 4 161, 1948
Wilson, J N V-Y Correction for Varus Deformity of the Fifth Toe, Brit J Surg 41 133-135, 1953

Hallux Valgus

Bernard, L End Results in Bunion Surgery, Ann Surg 91 937-940, June, 1930
Brandes, Max Zur operativen Therapie des Hallux valgus, Zentralbl Chir , No 39, 1929
Brookes, T P Hallux Valgus Postoperative Physical Therapy, 14th Annual Session American Congress of Physical Therapy, Kansas City, Mo , Sept , 1935
Cleveland, M , and Winant, E M An End-Result Study of the Keller Operation, J Bone & Joint Surg 32A 163-175, Jan , 1950
Durman, D C Metatarsus Primus Varus and Hallux Valgus, A M A Meeting Section on Orthopedic Surgery, Chicago, June 12, 1956
Durman, D C Metatarsus Primus Varus and Hallux Valgus, A M A Arch Surg 74 128, Dec , 1956
Galland, W I , and Jordan, H Hallux Valgus, Surg Gynec & Obst 66 95-99, Jan , 1938
Girdlestone, G R , and Spooner, H J A New Operation for Hallux Valgus and Hallux Rigidus, J Bone & Joint Surg 19 29-35, Jan , 1937
Haines, R W , and McDougall, A Anatomy of Hallux Valgus, J Bone & Joint Surg 36B 272-293, May, 1954
Hardy, R H , and Clapham, J C Observations on Hallux Valgus, J Bone & Joint Surg 33B 376-391, Aug , 1951
Hawkins, T B , Mitchell, C L , and Hedrick, D W Correction of Hallux Valgus by Metatarsal Osteotomy, J Bone & Joint Surg 27 387-394, July, 1945
Hueter, C Klinik der Gelenkkrankheiten mit Einschluss der Orthopadie Part II Specielle Pathologie der Gelenkkrankheiten der Extremitaten, ed 2, Leipzig, 1877, F C W Vogel, A 6-10
Joplin, R J Sling Procedure for Correction of Splay Foot, Metatarsus Primus Varus, and Hallux Valgus, J Bone & Joint Surg 32A 779-785, Oct , 1950
Joseph, J Range of Movement of Great Toe in Men, J Bone & Joint Surg 36B 450, Aug , 1954
Kaplan, E B The Tibialis Posterior Muscle in Relation to Hallux Valgus, Bull Hosp Joint Dis 16 88-93, April, 1955
Keller, W L Surgical Treatment of Bunions and Hallux Valgus, New York M J 80 741-742, Oct 15, 1904
Keller, W L Further Observations on Surgical Treatment of Hallux Valgus and Bunions, New York M J 95 696-698, April 6, 1912
Kleinberg, S Operative Cure of Hallux Valgus and Bunions, Am J Surg 95 75, Jan , 1932
Kreuscher, P H , and Kelikian, H Hallux Valgus (Bunion), Illinois M J 67 453-456, May, 1935
Levine, M A An Operative Technic for Hallux Valgus, J Bone & Joint Surg 20 923-925, Oct , 1938
Mayo, C H Surgical Treatment of Bunion, Ann Surg 48 300-302, Aug , 1908
Mayo, C H The Surgical Treatment of Bunions, Minnesota M J 3 326-330, July, 1920

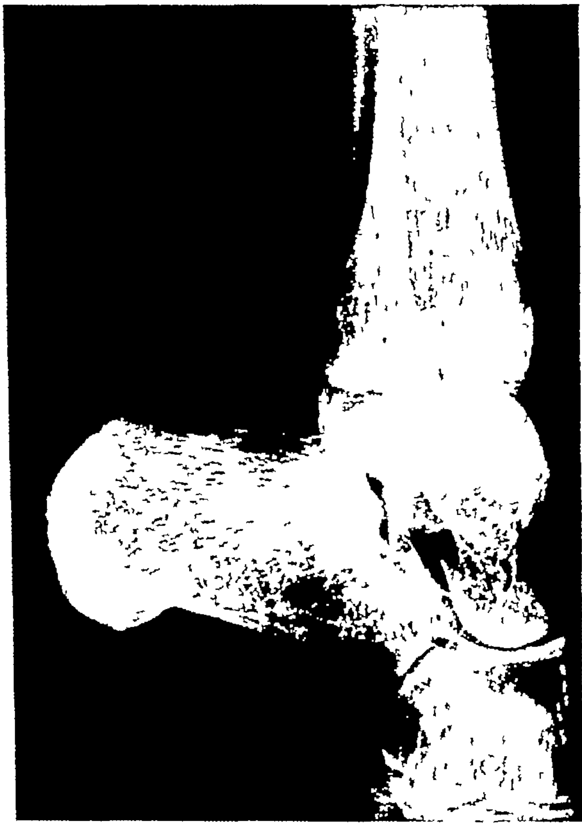


Fig 357

Fig 358

Fig 357 —Paralytic drop foot Note that forefoot, beginning at navicular, is in direct line with tibia

Fig 358 —Same case as shown in Fig 357, after insertion of Gill's posterior bone block

Some surgeons advise triple arthrodesis I prefer to use Gill's posterior bone block procedure which consists of excision of a triangular section from the superoposterior part of the calcaneus, followed by elevating the cartilaginous plate from the posterior part of the talus and wedging in the triangular section of bone Two weeks postoperatively, with the foot dorsiflexed as much as possible, the foot is immobilized for about eight weeks in a plaster walking cast

REFERENCES

Morton's Syndrome

Harris, R I, and Beath, T The Short First Metatarsal, J Bone & Joint Surg 31A 553-565, July, 1949

Hawkes, O A M On Relative Lengths of First and Second Toes of Human Foot From Point of View of Anatomy and Heredity, J Genetics 3 249, 1914

Jones, F W Structure and Function as Seen in the Foot, Baltimore, 1944, Williams & Wilkins Co

Jones, Robert Notes on Military Orthopedics, London, 1917, Cassell & Co , Ltd

Morton, D J The Human Foot, New York, 1935, Columbia University Press

Morton, D J Human Locomotion and Body Form, Baltimore, 1952, Williams & Wilkins Co , pp 93-125

Wolff, Julius Das Gesetz der Transformation der inneren Architektur der Knochen bei pathologischen Veranderungen der ausseren Knochenform, Berlin, 1884, Reichsdruckerei

Hammertoe

Borg, I Operation for Hammer-Toe, Acta chir scandinav 100 619-625, 1950

Forrester-Brown, M F Tendon Transplantation for Clawing of the Great Toe, J Bone & Joint Surg 22 1, Jan , 1938

Ilfeld, F W Pes Planus, J A M A 124 281, Jan 29, 1944
Jack, E A Naviculo-Cuneiform Fusion in Treatment of Flatfoot, J Bone & Joint Surg 35B 75-82, Feb, 1953
Jones, Robert Peroneal Spasm and Its Treatment, Report of Meeting of Liverpool Medical Institution, April 22, 1897, Liverpool Med Chir J 17 442, April 22, 1897
Jones, Robert The Soldier's Foot and Treatment of Common Deformities of Foot, Brit M J 1 709, 1916
Kidner, F C Prehallux (Accessory Scaphoid) in Its Relation to Flatfoot, J Bone & Joint Surg 11 831, 1929
Lapidus, P W Spastic Flatfoot, J Bone & Joint Surg 28 126-136, Jan, 1946
L'Episcopo, J, and Sabatelle, P E The Hoke Operation for Flat Feet, J Bone & Joint Surg 21 92, Jan, 1939
Lewin, Philip The Foot and Ankle, ed 3, Philadelphia, 1947, Lea & Febiger
Merryweather, R Spastic Valgus of Foot, Proc Roy Soc Med 48 103 1955
Schoolfield, B L Operative Treatment of Flatfoot, Surg Gynec & Obst 94 136, Feb, 1952
Webster, F S, and Roberts, W M Tarsal Anomalies and Peroneal Spastic Flatfoot, J A M A 146·1099, July 21, 1951
Weil, F Ueber Erfahrungen an operativbehandelten kontrakten Plattfussen, Ztschr Orthop 86·204, 1955
White, J W Congenital Flatfoot, J Bone & Joint Surg 22 547, July, 1940
Zadek, I Transverse-Wedge Arthrodesis for Relief of Pain in Rigid Flatfoot, J Bone & Joint Surg 27.453, April, 1935

Clawfoot

Alvik, Ivar Operative Treatment of Pes Cavus, Acta orthop scandinav 23 137-141, 1953 (abstracted, Surg Gynec & Obst 99 3, Sept, 1954)
Brockway, Alvia Surgical Correction of Talipes Cavus Deformities, J Bone & Joint Surg 22 81-91, Jan, 1940
Cole, W H Treatment of Claw-Foot, J Bone & Joint Surg 22:895-908, Oct, 1940
Garceau, G J, and Brahms, M A Preliminary Study of Selected Plantar-Muscle Denervation for Pes Cavus, J Bone & Joint Surg 38A 553, June, 1956
Hibbs, R A An Operation for Clawfoot, J A M A 73 1583, Nov 22, 1919
Kochs, J Laminectomy Followed by Spontaneous Correction of Foot Deformity, Accompanying Occult Spina Bifida, Munchen med Wchnschr 74 1877-1879, Nov, 1927
Saunders, J T Etiology and Treatment of Clawfoot, Arch Surg 30 180, Feb, 1935
Steindler, Arthur Operative Treatment of Pes Cavus, Surg Gynec & Obst 24 612, 1917

Metatarsus Adductus

Gill, A B An Operation to Make a Posterior Bone Block at the Ankle to Limb Foot Drop, J Bone & Joint Surg 15 166-170, Jan, 1933
Kite, J H Congenital Metatarsus Varus, Report of 300 Cases, J Bone & Joint Surg 32A 499-506, July, 1950
McCormick, D W, and Blount, W P Metatarsus Adductovarus ("Skewfoot"), J A M A 141 449-452, Oct 15, 1949
Speed, J S, and Knight, R A Campbell's Operative Orthopedics, ed 3, vol II, St Louis, 1956, The C V Mosby Co, p 2019

McBride, E D A Conservative Operation for Bunion, J Bone & Joint Surg 10 735-739, Oct, 1928
McBride, E D A Conservative Operation for "Bunions," J A M A 105.1164-1168, Oct 12, 1935
McBride, E D Hallux Valgus, Bunion Deformity Its Treatment in Mild, Moderate, and Severe Stages, J Internat. Coll Surg 21 99-105, Jan, 1954
McElvenny, R T A Study of Hallux Valgus Its Cause and Operative Management, Quart Bull Northwestern Univ M School 18 286-297, 1944
McElvenny, R T, and Thompson, F R A Clinical Study of 100 Patients Subjected to Simple Exostosectomy for the Relief of Bunion Pain, J Bone & Joint Surg 22 942-951, Oct, 1940
Morton, D J The Human Foot, New York, 1935, Columbia University Press
Robinson, H A Bunion, Its Cause and Cure, Internat Clin 2 64, 1927
Schein, A J The Keller Operation, Partial Phalangectomy in Hallux Valgus and Hallux Rigidus Report of 55 Operations in 32 Cases, Surgery 7 342, March, 1940
Silver, David Operative Treatment of Hallux Valgus, J Bone & Joint Surg 5 225-232, April, 1923
Soresi, A L Radical Cure of Hallux Valgus (Bunion), Surg Gynec & Obst 52 776-777, March, 1931
Stanley, L L, and Breck, L W Bunions, J Bone & Joint Surg 17 961-964, Oct, 1935
Stein, H C Hallux Valgus, Surg Gynec & Obst 66 889-898, May, 1938
Truslow, W Metatarsus Primus Varus or Hallux Valgus, J Bone & Joint Surg 7 98-108, 1925

Hallux Varus

Haas, S L An Operation for the Correction of Hallux Varus, J Bone & Joint Surg 20 705-708, July, 1938
Horwitz, M T Unusual Hallux Varus Deformity and Its Surgical Correction, J Bone & Joint Surg 3 828-830, July, 1937
Sloane, David Congenital Hallux Varus, J Bone & Joint Surg 27 209-211, Jan, 1935

Hallux Flexus

Lapidus, P W "Dorsal Bunion" Its Mechanics and Operative Correction, J Bone & Joint Surg 22 627-637, July, 1940

Hallux Rigidus

Bingold, A C, and Collins, D H Hallux Rigidus, J Bone & Joint Surg 32B 214-222, May, 1950
Mau, C Disease Picture of Hallux Rigidus, Munchen med Wchnschr 75 1193, July 13, 1928
Miller, L F, and Arendt, J Deformity of the First Metatarsal Head Due to Faulty Foot Mechanics, J Bone & Joint Surg 22 349-353, April, 1940

Second Metatarsophalangeal Dislocations

Branch, H E Pathological Dislocation of the Second Toe, J Bone & Joint Surg 19 978-983, Oct., 1937
DuVries, H L Dislocation of Toe, J A M A 160 729, Feb, 1956

Flatfoot (Pes Planus)

Bingham, R Painful Feet, J A M A 124 283, Jan 29, 1944
Blockey, N J Peroneal Spastic Valgus of Foot, Proc Roy Soc Med 48 103, 1955
Butte, F L Navicular-Cuneiform Arthrodesis for Flatfoot, End-Result Study, J Bone & Joint Surg 19 496-502, April, 1937
Crego, C H, and Ford, L T An End-Result Study of Various Operative Procedures for Correcting Flat Feet in Children, J Bone & Joint Surg 34 183, Jan, 1952
Dively, R L Foot Imbalance, J A M A 103 1510, Nov 17, 1934
Graham, J Treatment of Weak Feet, Illinois M J 75 425-435, May, 1939
Harris, R I, and Beath, T Hypermobile Flatfoot With Short Tendo Achillis, J Bone & Joint Surg 30A 116, Jan, 1948
Harris, R I, and Beath T Etiology of Peroneal Spastic Flatfoot, J Bone & Joint Surg 30B 624-634, Nov, 1948
Hauser, E D W Diseases of the Foot, ed 2, Philadelphia, 1950, W B Saunders Co
Hoke, M Operation for Stabilizing Paralytic Feet, Am J Orthop Surg 3 494, 1921
Hoke, M Operation for Correction of Extremely Relaxed Flat Feet, J Bone & Joint Surg 13 773-783, Oct, 1931

Ilfeld, F W Pes Planus, J A M A 124 281, Jan 29, 1944
Jack, E A Naviculo-Cuneiform Fusion in Treatment of Flatfoot J Bone & Joint Surg 35B 75-82, Feb , 1953
Jones, Robert Peroneal Spasm and Its Treatment, Report of Meeting of Liverpool Medical Institution, April 22, 1897, Liverpool Med Chir J 17 442, April 22, 1897
Jones, Robert The Soldier's Foot and Treatment of Common Deformities of Foot, Brit M J 1 709, 1916
Kidner, F C Prehallux (Accessory Scaphoid) in Its Relation to Flatfoot, J Bone & Joint Surg 11 831, 1929
Lapidus, P W Spastic Flatfoot, J Bone & Joint Surg 28 126-136, Jan , 1946
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Lewin, Philip The Foot and Ankle, ed 3, Philadelphia, 1947, Lea & Febiger
Merryweather, R : Spastic Valgus of Foot, Proc Roy Soc Med 48 103 1955
Schoolfield, B L Operative Treatment of Flatfoot, Surg Gynec & Obst 94 136, Feb , 1952
Webster, F S , and Roberts, W M Tarsal Anomalies and Peroneal Spastic Flatfoot, J A M A 146 1099, July 21, 1951
Weil, F Ueber Erfahrungen an operativbehandelten kontrakten Plattfussen, Ztschr Orthop 86 204, 1955
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Kochs, J Laminectomy Followed by Spontaneous Correction of Foot Deformity, Accompanying Occult Spina Bifida, Munchen med Wchnschr 74 1877-1879, Nov , 1927
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Steindler, Arthur Operative Treatment of Pes Cavus, Surg Gynec & Obst 24 612, 1917

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McCormick, D W , and Blount, W P Metatarsus Adductovarus ("Skewfoot"), J A M A 141 449-452, Oct 15, 1949
Speed, J S , and Knight, R A Campbell's Operative Orthopedics, ed 3, vol II, St Louis, 1956, The C V Mosby Co , p 2019

CHAPTER SEVENTEEN

Anomalies and Congenital Defects*

ANOMALOUS MALFORMATIONS MAY BE CONGENITAL OR ATAVISTIC THEY ARE COMMONER in the foot than in any other part of the body, although they may appear in the foot only as one of a group of multiple abnormalities throughout the body (Farmer, 1948) Atavistic defects are inherited characteristics from remote rather than immediate ancestry Examples pertinent to the present text are tarsal coalition, congenital vertical talus in association with congenital flatfeet (Osmond-Clarke, 1956, Lloyd-Roberts and Spence, 1958), accessory bones and accessory parts, absence of a part or of an entire bone, or even an entire foot, syndactylism (web toes), polydactylism (extra toes), microdactylism (midget toes), macrodactylism or megalodactylism (giant toes), and limitless deviations from the normal, even a unique case of pedal anomaly of a foot, a supernumerary or rudimentary foot (Hayden and Murray, 1956), or the rare developmental anomaly of the lateral malleolus, mistakenly diagnosed as fracture (Bjornson, 1956)

The reasons for malformations of the foot are still conjectural Congenital defects and deformities are thought to be the result of embryologic maldevelopment. Ordinary frequent examples† are hammertoe, overlapping toe, hallux varus, metatarsus adductus, and the diverse talipes

Davis and Hatt (1955) have contributed a necessary article regarding roentgenographic techniques and roentgenologic analyses of congenital abnormalities of the foot.

THE INFANT FOOT

At birth, the foot may appear deformed because it still retains some of the primitive features of the primates, such as appearing as a pes adductus A longi-

*Spina bifida, in relation to the lower extremities, and spondylolisthesis are discussed in Chapter 13, Diseases of Nerves

†Hammertoe, overlapping toe, hallux varus, and congenital metatarsus adductus are discussed in Chapter 16, Static Deformities

tudinal arch is lacking and the foot appears flat. Except for obvious deformities, however, such initial deficiencies need not cause concern. The foot may continue to appear adducted and flat until the age of 2 years, because, until then, development is insufficient to carry out the function of weight bearing and propulsion. During the first two years the infant's muscles develop by his kicking, twisting, and crawling. The age at which children begin to stand or walk varies within widely normal limits. A child should not be urged to stand or walk until he is naturally ready. Between the ages of 2 and 4 years, a longitudinal arch develops, and the child assumes a normal gait. By his running and jumping, his skeletal structure of the foot and leg develops further, but it is during this period that the normal attitude of the foot may deviate, and such disorders may be noticed as flatfoot accompanied by abduction of the forefoot and pes adductus (pigeon toe). At this stage, correction is possible in most instances by shoe appliances or padding and wedging of the shoe. In extreme cases, plaster casts or surgical intervention may be required.

Gross congenital deformities and anomalies of the foot in infancy and childhood are self-evident, but some types are unnoticed until static deformities develop. Shands and Wentz (1953) reviewed 4,230 cases of disorders of the foot in children treated at the Alfred I du Pont Institute, and selected for study those in which roentgenograms had been taken, 850 in all. Of the selected cases, they found that 231, or 27 per cent, showed significant congenital anomalies.

The prognosis in cases of congenital deformities of the foot is in direct relation to early diagnosis and treatment.

THE PRE-ADOLESCENT FOOT

Foot problems during the pre-adolescent period differ from those during adulthood only in the changes in tissue through growth and activities normal to the growing child. Ossification of the epiphysis takes place during adolescence but at varying years. The degree of ossification of the different bones of the foot and ankle varies according to the year in which ossification takes place. Slight injuries or wearing of poorly fitted shoes predispose the child to osteochondritis or fracture of the epiphyseal plate. Healing capability of the child and adolescent is superior to that of the adult, although children have a high potential of injuries incurred at play.

AGE OF OSSIFICATION CENTERS

The only bones seen in roentgenograms of the foot at birth are the diaphysis of the phalanges and metatarsals and the nuclei of the calcaneus, talus, and sometimes the cuboid. The nuclei of the calcaneus and talus show the typical shape of the bone at birth. The calcaneus is formed by the fusion of two nuclei which are present in the early embryonic stage.

During the first few months the nucleus of the cuboid begins to ossify, this is followed by the third cuneiform during the sixth postnatal month. At the beginning of the second year, the nuclei of the first cuneiform and of the epiphysis of the phalanges are observable. Toward the end of the second year,

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Fig 360

Fig 361

Fig 360 —Congenital talipes equinovarus in an infant
Fig 361 —Left, Normal foot Right, Congenital talipes varus in an infant

weight is borne by the shaft and base of the fifth metatarsal (Fig 362) The types vary in degree and may appear singly or in combination in the following order of frequency talipes valgus, talipes equinus, talipes equinovarus, talipes equinovalgus, talipes calcaneus, talipes calcaneovarus, and talipes calcaneovalgus

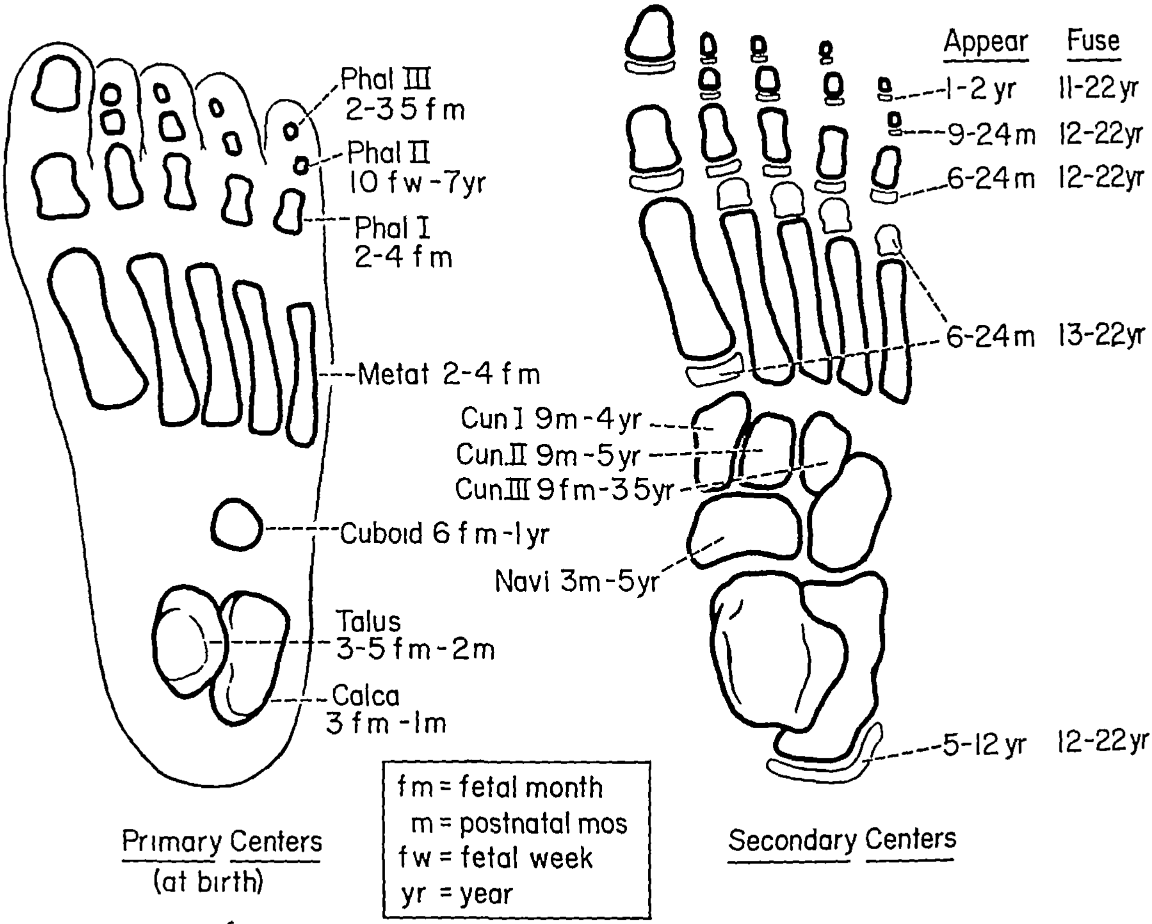


Fig 359 —Ossification centers of foot (Modified from Caffey)

all the tarsal nuclei may be present as well as the epiphysis of the metatarsals and phalanges In the male, the nucleus of the middle cuneiform may not appear until the end of the fourth year, and the nucleus of the navicular may not be seen until the fourth year (Fig 359)

CONGENITAL TALIPES

The most prevalent and most important congenital deformity of the foot is talipes (clubfoot), which is present at birth, it is often neglected during infancy when it can be corrected

Talipes is derived from *talus* (astragalus) and *pes* (foot) The basic types of congenital talipes are equinus, calcaneus, valgus, and varus In the *equinus* type (Fig 360), the foot is held in plantar flexion, calf muscles are contracted, and the toes are contracted and hammered (clawtoes) When the child begins to walk, he bears weight only on the ball of the foot In the *calcaneus* type, the anterior muscles are contracted, the foot is held in dorsiflexion, and weight is borne only by the heel In *valgus* talipes, the foot is abducted and everted, the peroneal muscles are contracted, and weight is borne by the shaft of the first metatarsal, first cuneiform, and navicular bones A *varus* talipes (Fig 361) means that the foot is adducted and inverted The tibialis anticus is contracted and

servative treatment in infancy and early childhood, however, is effective in most instances

The basic step in the correction of a clubfoot in early childhood is to place all the components of the deformity in an exact opposite or overcorrected position through continuous retentive leverage by means of splints or casts until a normal attitude of the foot has been attained Some surgeons prefer splints (Browne, 1933, Thomson, 1942, Blumenfeld and associates, 1946), others prefer casts (Kite, 1932, 1939, 1957) Relapsed or only partly corrected cases have followed both procedures Perhaps most failures are the result of hasty evaluation of the deformity or of inadequate subsequent treatment

The Denis Browne splint has been modified so as to protect the normal foot in unilateral cases. This has been fully described by Thomson (1955)

White (1951) suggested that skin-tight casts with sponge-rubber pads over the three pressure points, to protect against pressures sores, are preferable to other methods, because they "maintain, without wedging, a constant corrective force for two weeks or more" The advantage in not requiring the wedging between plaster changes is in reducing the number of hospital visits

Regardless of the procedure employed, it is important to study the clubfoot in two parts, the forefoot and the hindfoot, each to be evaluated separately The forefoot deformity should be corrected first and the hindfoot weeks later Unless the adduction deformity is corrected first and the flexion deformity weeks later, a *rocker foot* results The head of the calcaneus must be kept away from under the neck of the talus Failure to correct the forefoot tends to recurrence (Bechtol and Mossman, 1950) Abnormal insertions and attachments of the opponent adductors and abductors of the forefoot may produce an imbalance in their function and cause recurrence of the deformity, as has been pointed out by Scherb (1930), who explained how abnormality of tendinous attachment, particularly of the peroneus brevis to the extensor brevis digitorum, causes recurrence of the adduction type of deformity Garceau and Manning (1947) have shown how abnormal attachments of the tibialis anterior tendon lead to recurrence

Weight bearing should be limited while a corrective device is worn in order to cause some atrophy of the bones which makes correction easier

Kite applies a cast from the toes to the midthigh The knee is flexed in a 90 degree angle and the foot held in mild correction without undue strain on the deformity It takes longer to obtain full correction by this method but better results are obtained The cast is changed once or twice a week When the deformity has been completely corrected, a retentive cast is applied for several months The retentive cast should also be changed every few weeks

The Denis Browne splint works on the principle that "it is possible to control the position of one foot by the other" In the infant this is accomplished by bandaging and strapping both feet to metal foot plates, plates are fastened to a horizontal cross bar (Fig 363) The foot must be protected from skin irritation and pressure points by sufficient padding The strapping and bandaging are changed every five to eight days Correction of the deformity takes place in from six to eight weeks, however, the foot must be held in a normal position

Etiology.—It may be assumed that the bases of all current theories are contributory in the causation of anomalies and congenital defects

Heredity—Heredity exerts an influence through one or both parents. Two or more children with clubfoot may be born of the same mother, however, as a rule, nothing bearing on the deformity is evident in the family or personal history

Mechanical—Mechanical possibilities in causation are intrauterine pressure on the fetus, through compression of the uterus or by another part of the fetus, or the abnormal position or entanglement of the umbilical cord, or of amniotic bands Interlocking of the fetal feet also has been suggested as a cause of the deformity

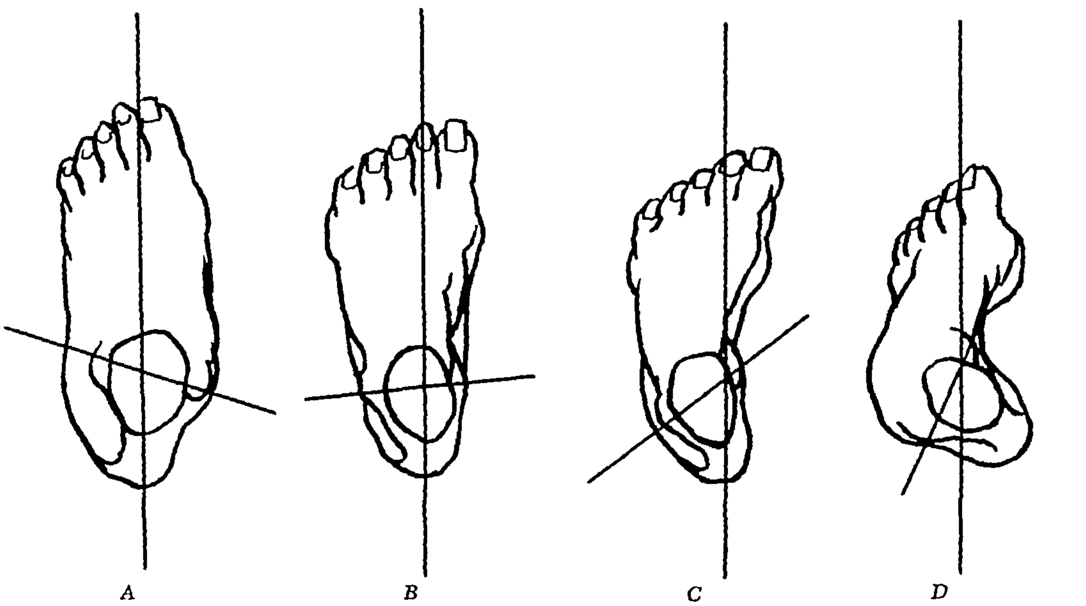


Fig 362—Schematic drawing of normal foot and three types of talipes deformity *A,* Talipes valgus *B,* Normal *C,* Metatarsus adductus *D,* Talipes varus

Musculo-Nervous Alterations—An alteration in muscle balance, with or without central nervous lesion, during intrauterine life, may be followed by contraction of certain muscles causing the deformity This view was supported by many of the early writers, although some recent writers also hold the view Bechtol and Mossman (1950) believe that clubfoot is caused by a lack of balance of opponent muscles, Stewart (1951) believes that abnormal differentiation of tendons may be instrumental Knight (1954) studied the postural habits of infants and children during sleep, while sitting, and at play, and concluded that when those habits are faulty, they are frequently indirectly the cause of developmental deformities of the foot

Arrested Development—When the physiologic position of the feet in the sixth or seventh fetal week has the sole turned inward, that position is permanently maintained and thus causes the deformity, according to the theory of developmental arrest

Treatment of Congenital Clubfoot in Early Childhood—Numerous operative procedures have been proposed for the treatment of static clubfoot Con-

Beath, 1948) Harris and Beath in 1948 first published the rationale of their theory of tarsal coalition as the cause of rigid flatfoot. O'Rahilly (1953) surveyed the records of tarsal and carpal anomalies Pfitzner (1896) described nine anatomic specimens of partial or complete bridging of the talus and calcaneus and fifteen specimens of calcaneonavicular bridging Slomann (1921) reported the first clinical case of calcaneonavicular fusion Since then, many reports have been made on the clinical aspects of the anomaly *Symphalangism* was Harvey Cushing's term for "congenital absence or fusion of interphalangeal joints" (Austin, 1951). Harris and Beath (1948) suggested that a large number of spastic flatfeet were due to tarsal bridges This concept, which they explored studiously, has since been supported by many investigators, notably Webster and Roberts (1951), Jack (1954), de Marchi and co-workers (1955), and Waugh (1957) The types

Fig 365 —Synostosis of talocalcaneal articulation Arrow points to bar

of fusion reported most are talocalcaneal (Fig 365), talonavicular, sustentaculum-navicular, and calcaneocuboid Fusion of the other tarsal bones undoubtedly occurs, preventing any movement of that joint and often resulting in static foot deformities When fusion occurs, the anomaly may appear as a narrow bar or partial or complete fusion of two bones, it may be osseous *(synostosis)*, cartilaginous *(synchondrosis)*, or fibrous *(syndesmosis)* The cause is an error of differentiation of the mesenchyma during formation of the tarsal bones Such an error may lead to formation of a small accessory bone or to complete fusion of two adjacent bones (Jack, 1954)

Symptoms.—Rigid tarsal joints due to tarsal coalition probably exist from birth, however, in early childhood, the condition causes few, if any, symptoms Foot discomfort is likely to appear first during adolescence or early adult life Sometimes symptoms do not appear until middle life, and then trauma may be the trigger mechanism A single injury, such as a fall or jump, or repeated daily

for many months, even as long as a year, to prevent recurrence When the child can be fitted with a firm high-top shoe, the sole of the shoe can be fastened to the foot plate (Fig. 364) and the appliance worn night and day The splint may be removed occasionally for a few hours In mild cases, firm high shoes may be worn during the day and splints at night In severe cases, the splint should be worn continuously, with only short rest periods, for a year or longer

Manipulations and repeated tenotomies and capsulotomies are useless (Chollett, 1933) Wedge osteotomies and arthrodesis of some of the tarsal joints, with or without tendon transference, must be resorted to when clubfoot resists correction by more conservative measures No two cases are ever precisely alike

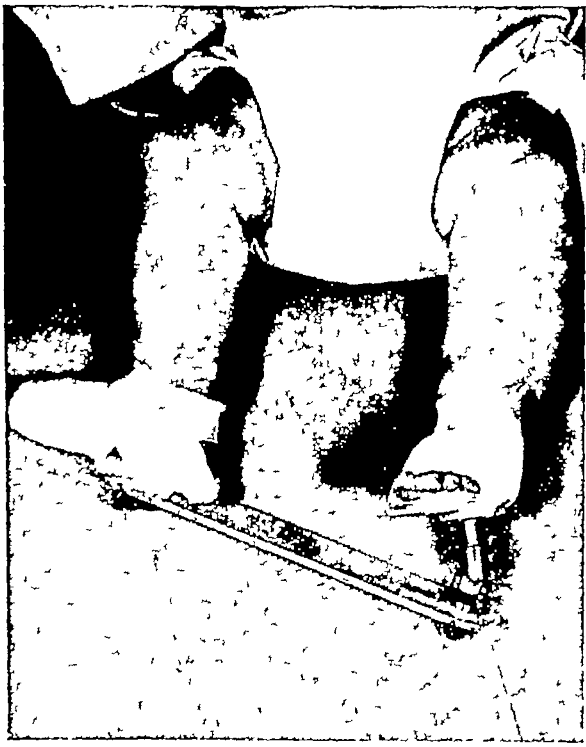


Fig 363

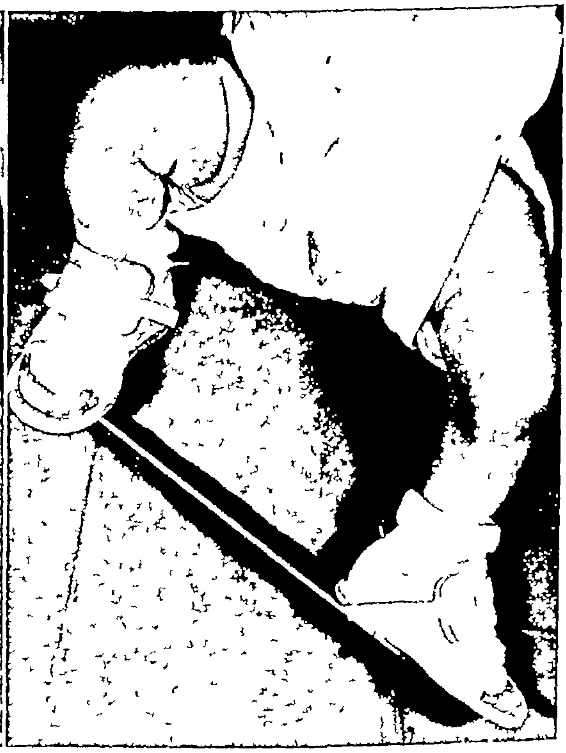


Fig 364

Fig 363 —Denis Browne splint applied and held with adhesive strapping (Courtesy Dr S A Thomson)
Fig 364 —Denis Browne splint applied, with shoes (Courtesy Dr S A Thomson)

The plan of operation must be studied carefully for application to each particular case The basic osteotomies and arthrodeses are numerous but well covered in available publications solely concerned with the varieties of talipes Clawfoot, flatfoot, drop foot, and metatarsus adductus, which are deformities primarily of the forefoot and therefore within the limitations of this text, are discussed in Chapter 16, Static Deformities

TARSAL COALITION

Anomalous fusion of two or more tarsal bones, *tarsal coalition*, has long been recognized, a specimen even having been prepared by John Hunter (Harris and

stresses from occupations requiring standing for long periods, or marching may give rise to symptoms. Different types of anomalies produce both similar and varied symptoms and deformities. The coalition may be painful without concomitant deformity or rigidity of all the subtalar joints. Pain is due to a compensatory excessive strain on the other tarsal joints because of the rigidity of the fused joints, often resulting in spasm of the leg muscles, especially the peroneal group. Rigidity of the subtalar joints is soon noticed when an attempt is made to invert the foot. In flaccid flatfoot, there is a wide range of movement in the tarsus, whereas in the rigid foot, there is little if any movement of the subtalar joints.

Metatarsal coalition (synostosis) (Figs. 366 and 367) only becomes symptomatic when accompanied by severe deformity.

Talocalcaneal, talonavicular, and calcaneonavicular fusions often become symptomatic. They are a frequent, perhaps the commonest, cause of *rigid flatfoot* (Harris, 1955) or *peroneal spastic flatfoot* (Webster and Roberts, 1951). Jack (1954) reviewed sixty-eight cases of rigid flatfoot, thirty of them his own cases, in which twenty-seven showed talocalcaneal bars, twenty-three calcaneonavicular bars, and two, both types; sixteen were without bone abnormality. Harris (1955), who has contributed notably to disorders of the feet, has also discussed the problem of failure to demonstrate roentgenographically talocalcaneal bridges in cases of rigid valgus foot. He ascribed the obstruction to the roentgen beam to "bone blocking" and in 1955 was still seeking a satisfactory roentgenographic technique.

Diagnosis.—Bony coalitions can be demonstrated roentgenographically, however, more than the usual routine views are often necessary to disclose the anomaly. Vaughan and Segal (1953) advised a lateral and external oblique (eversion) view of the ankle as most valuable in demonstrating the calcaneonavicular coalition. The subtalar joint is best visualized by a lateral and modified plantar view, as described by Sante (1944). Cartilaginous or fibrous union can be suggested only by clinical observations.

Treatment.—Asymptomatic cases should be treated expectantly. Mild cases call for relief by shoe wedging or inlay supports. In moderately severe cases, especially those in which there is some mobility in the subtalar articulations and in which pain is localized about the bridge, excision of the bar relieves symptoms. In long-standing serious cases of completely rigid flatfoot with extreme eversion, a stabilization operation is necessary.

ACCESSORY BONES

The many accessory bones of the foot rarely become symptomatic. The os trigonum and the prehallux are exceptions. These two anomalous bones often become disabling.

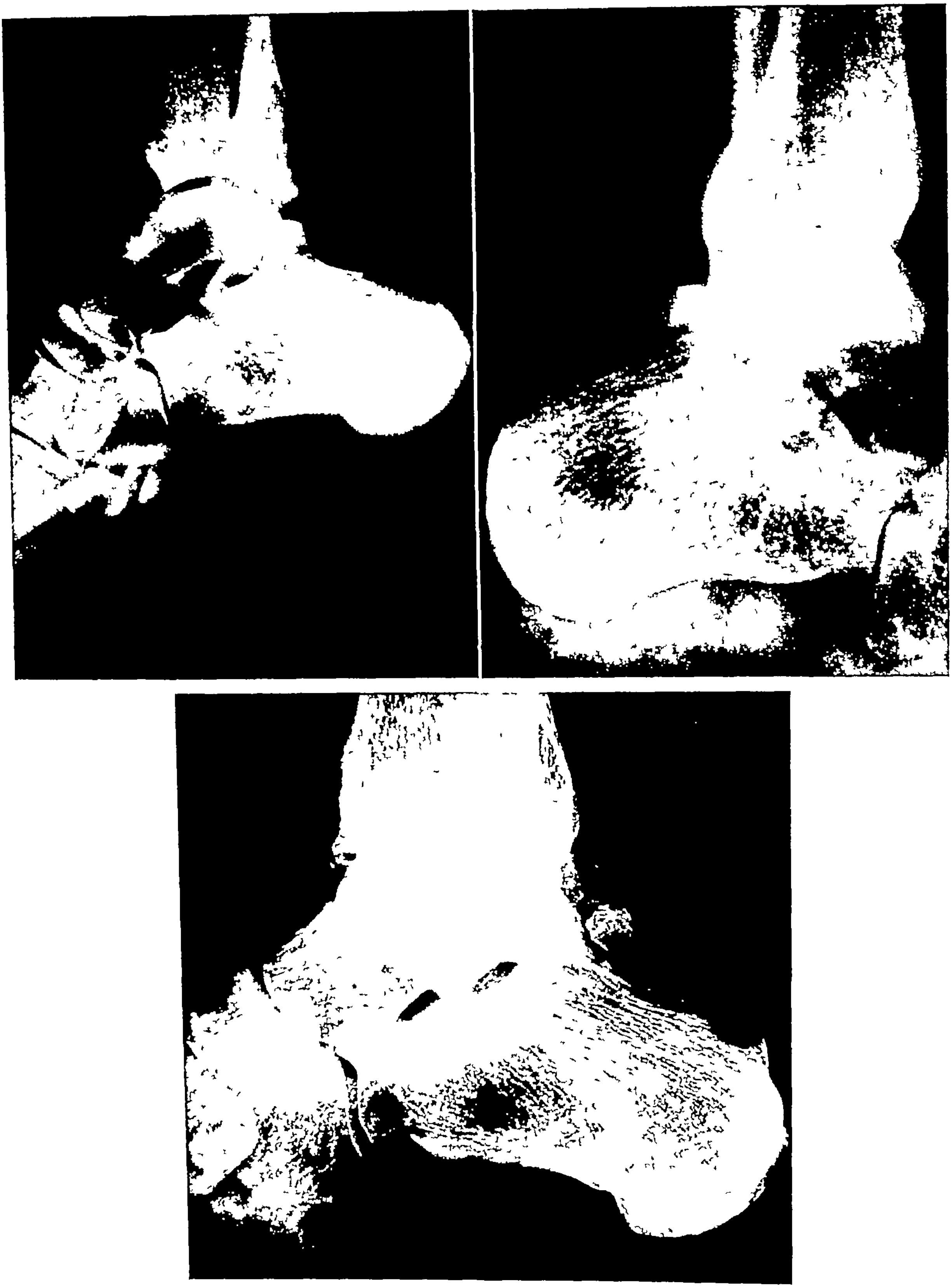
Os Trigonum

The os trigonum varies greatly in size and shape and, appearing at the posterior process of the talus, may be an actual part of the body of the talus (Fig.

Fig 366 —Congenital synostosis of second and third metatarsals, asymptomatic

Fig 367 —Congenital synostosis of fourth and fifth metatarsals Plantar projection, shown at left, caused pain

A B



C

Fig 369 —*A*, Os trigonum attached to talus by cartilage plate Note os vesalianum at base of fifth metatarsal *B*, Os trigonum attached to talus by cartilaginous plate *C*, Os trigonum completely detached from body of talus

368), or, on the other hand, it may be a completely separate bone which may or may not adhere to the talus by a cartilaginous plate (Fig 369) Bizarro (1921) found from his studies that the ossicle appeared in seven of 100 roentgenograms of the feet The ossicles are usually asymptomatic and are detected only during routine roentgenography They have been mistaken for fracture of the posterior process of the talus McDougall (1955) presented evidence to show that the posterior process arises from separate ossification centers and that the ossicles appear between 8 and 11 years of age

The ossicles which are separated from the talus may become loose, producing pain on plantar flexion of the foot, whereas those which are part of the talus may be fractured

Fig 368 —Os trigonum as an actual part of posterior part of talus

The normal plantar flexion of the ankle joint is limited by the locking of the posterior tubercle of the talus against the posterior lip of the articular surface of the tibia (Fig 370) In an active person, the posterior lip of the talus strikes the posterior lip of the tibia hundreds of times, in athletes, it may strike with force That is why if the tubercle is large and attached to the body of the talus by a thin neck of bone or cartilage, it may become fractured or loosened

Symptoms—The symptom is pain in the retrocalcaneal space Pain is aggravated on walking and especially when the foot is in plantar flexion When pressure is applied with the thumb and index finger against the posterior lip of the talus, pain is sharp The onset of symptoms is gradual except in sudden fracture of an attached os trigonum The symptoms become progressively worse

Differential Diagnosis.—The condition must be differentiated from retrocalcaneal bursitis wherein the symptoms are acute, with swelling and tenderness over the retrocalcaneal space, more so always posteriorly and just above the

Procedure for Removal of Os Trigonum.—By the procedure for removal of the os trigonum outlined here, ambulation may begin in four days Healing is uneventful in most cases (Fig. 371)

1 Make a linear incision, about 8 cm. long, over the retrocalcaneal space just behind the lower end of the fibula

2 Retract the skin and make a similar incision in the fascia, deflect the margins The anterior margin of the fascia will be contiguous with the sheath of the peroneal tendons, which are readily retracted to expose the retrocalcaneal space

3 Denude the os trigonum of all attachments with small scissors The separated type of os trigonum can usually be brought out of the wound The adherent type may be amputated by means of a nasal saw or an osteotome

4 Smooth the cut surface with a rasp

5 Suture the fascia and skin in layers

6 If there is indication that the dead space in the wound should be occluded, it may be accomplished by placing a button over both sides of the retrocalcaneal space and passing a wire through the space and the holes of the button

Accessory Navicular (Tarsal Scaphoid)

Accessory navicular is a congenital anomaly wherein the tuberosity develops from a second center of ossification The disabling effects of this accessory bone is commoner than is generally realized In 1925 Geist reported a 14 per cent incidence of accessory navicular in supposedly normal feet There are two distinct types One is a typical, usually small, accessory bone without attachment to the body of the navicular but with a well-defined round or oval outline of demarcation (Fig 15) The other type is a definite part of the body of the navicular, but the tuberosity is separated by a fibrocartilaginous plate of irregular outline (Figs 372 to 374). The first type is a true *os tibiale externum* or *navicular secundarium* Because it hardly ever produces symptoms, it is unnecessary to dwell on it here The second type often has an elongated neck articulating with the tibial side of the head of the talus and fits the description of *prehallux* or *bifurcated navicular* It is likely to become symptomatic and may be mistaken for fracture of the neck of the navicular, therefore, the discussion concerns this type

Zadek (1926) studied fourteen cases of symptomatic accessory navicular In five, he observed roentgenographically definite fusion with the body of the navicular, in three, partial fusion, and in six, complete separation Later, in 1948, Zadek and Gold studied microscopically the structures connecting the accessory bone to the body of the navicular and recorded that they were variously composed of a soft tissue plate of hyaline cartilage, dense fibrocartilage, or both and sometimes showed ossification as well

The condition is an architectural defect which interferes with the structural stability of the foot. When a prehallux is present, the tendon of the tibialis posticus which normally inserts into the plantar surface of the tuberosity of the navicular, with some fibers extending to the three cuneiforms, may have most of its attachment inserted into the prehallux Kidner (1929) called attention to the

insertion of the tendo achillis instead of at the anterior part of the retrocalcaneal space The roentgenograms either do not show the os trigonum at all or they reveal only a rudimentary ossicle Symptomatic cases require surgical removal of the os trigonum

Fig 370

Fig 371

Fig 370 —Os trigonum causing pain on plantar flexion

Fig 371 —Same foot as shown in Fig 368 one year after removal of os trigonum, completely symptom-free

A B

Fig 374 — A, Large accessory navicular, extending under body of navicular, caused abduction of forefoot B, Two years postoperatively Note correction of forefoot abduction

altered line of the pull caused by the insertion of the attachment of the tendon of the tibialis posticus into the prehallux Instead of pulling the foot directly plantarwise as it normally does, the attachment of the tendon pulls backward and inward at an angle, thus adducting the foot This crowds the medial side of the talonavicular joint, causing pain. That is why pain is alleviated when the foot is forced in abduction (See Fracture of the Navicular Tuberosity, page 332)

The occurrence of these anomalies is related to the inconstant accessory bones of the foot which, in most instances, are only of academic interest, but from time to time such bones as the accessory navicular may produce symptoms requiring surgical intervention

Symptoms.—Symptomatic cases occur in early adulthood, especially in women Most of my thirty cases were in women The anomaly appears equally in the two sexes, but women's shoes undoubtedly cause symptoms As a rule, the anomaly is bilateral, but symptoms appear only in one foot Patients state that the protrusion was always present over the medial side of both navicular bones and that one foot gradually became progressively painful and swollen, even at rest Palpation of the navicular bone elicits sharp, deep pain, especially when the foot is adducted and inverted Roentgenograms verify the diagnosis

Treatment.—In early cases of moderate symptoms, conservative expectant measures relieve pain Such measures include a shoe that exerts little or no pres-

Fig 372 —*A*, Large accessory navicular, cartilaginous plate loosened and painful *B*, One year postoperatively

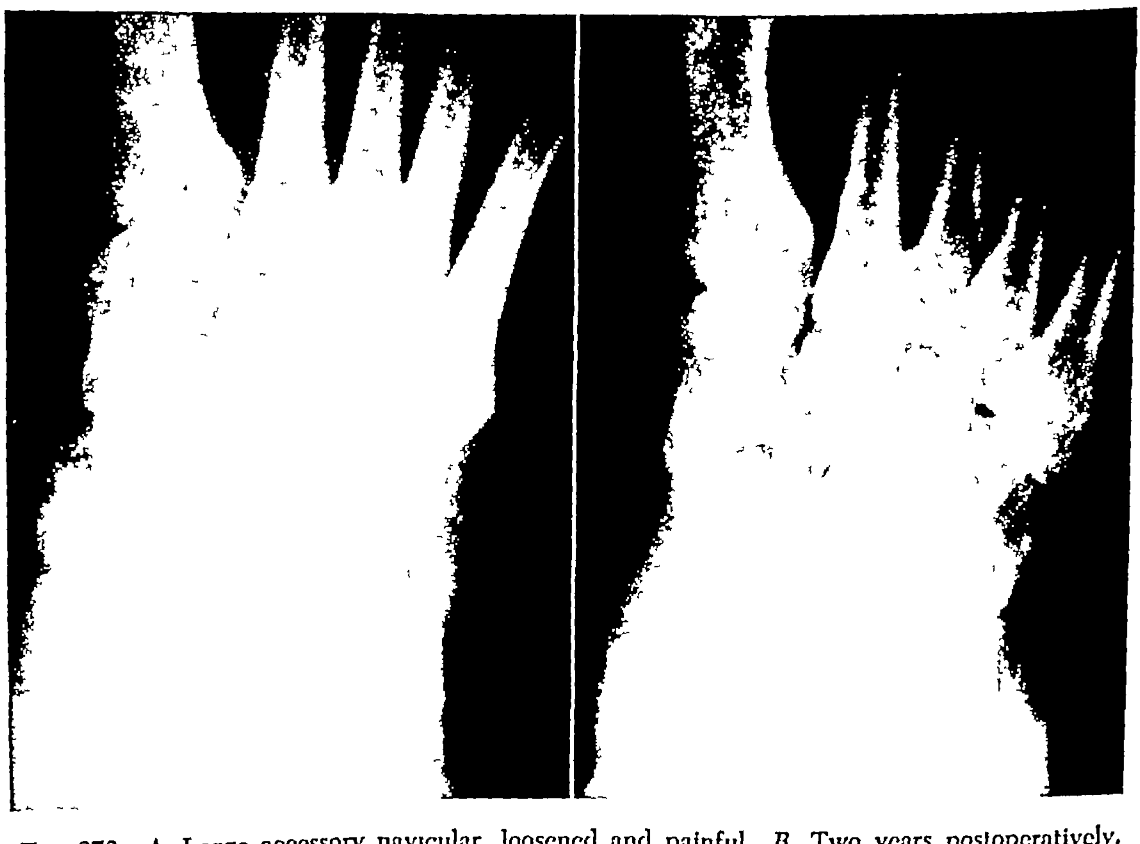


Fig 373 —*A*, Large accessory navicular, loosened and painful *B*, Two years postoperatively.

sure over the navicular, wedging of the shoe for added support under the talonavicular joint, strapping, and an elastic ankle support for partial immobilization of the joint The intractable case requires surgical intervention. DuVries' procedure is outlined.

1 Make a semielliptical incision, with the vertex dorsally, over the talonavicular joint, retract the medial skin margin to expose the bony prominence covered by the ligaments and fascia

2 Make a vertical incision in the fascial structures, extending from behind the neck of the talus to the medial side of the medial cuneiform, denude the fascia from the protruding bone and retract Manual motion of the accessory bone, independent of the body of the navicular, can be elicited at this stage.

3. Make an incision into the area where motion is felt to separate the tuberosity from the body of the navicular

4 Continue the incision between and around the bone fragment Sometimes bone forceps are needed to remove the accessory bone

5 Because the adjacent surface on the body of the navicular is rough and irregular, it should be smoothed and flattened with a rasp so as to be even with the neck of the talus (Figs 375 and 376)

6 Kidner (1933) suggested suturing the tendon of the tibialis posticus to the body of the navicular

7 Close the ligaments and skin in layers and apply a compression bandage

8 Weight bearing is not permitted for one week, at which time the sutures are removed and a walking cast is applied with the foot slightly adducted and inverted

9 Remove the cast in about five weeks, after which the patient wears a well-fitted Oxford with an extended counter for six months.

Accessory Medial Cuneiform (Os Paracuneiforme)

The rare accessory medial cuneiform presents a problem similar to that of an accessory navicular Morrison (1953) reviewed the publications and reported a case in which the patient required surgical intervention for relief of symptoms The procedure for removal is comparable to that described for removal of an accessory navicular

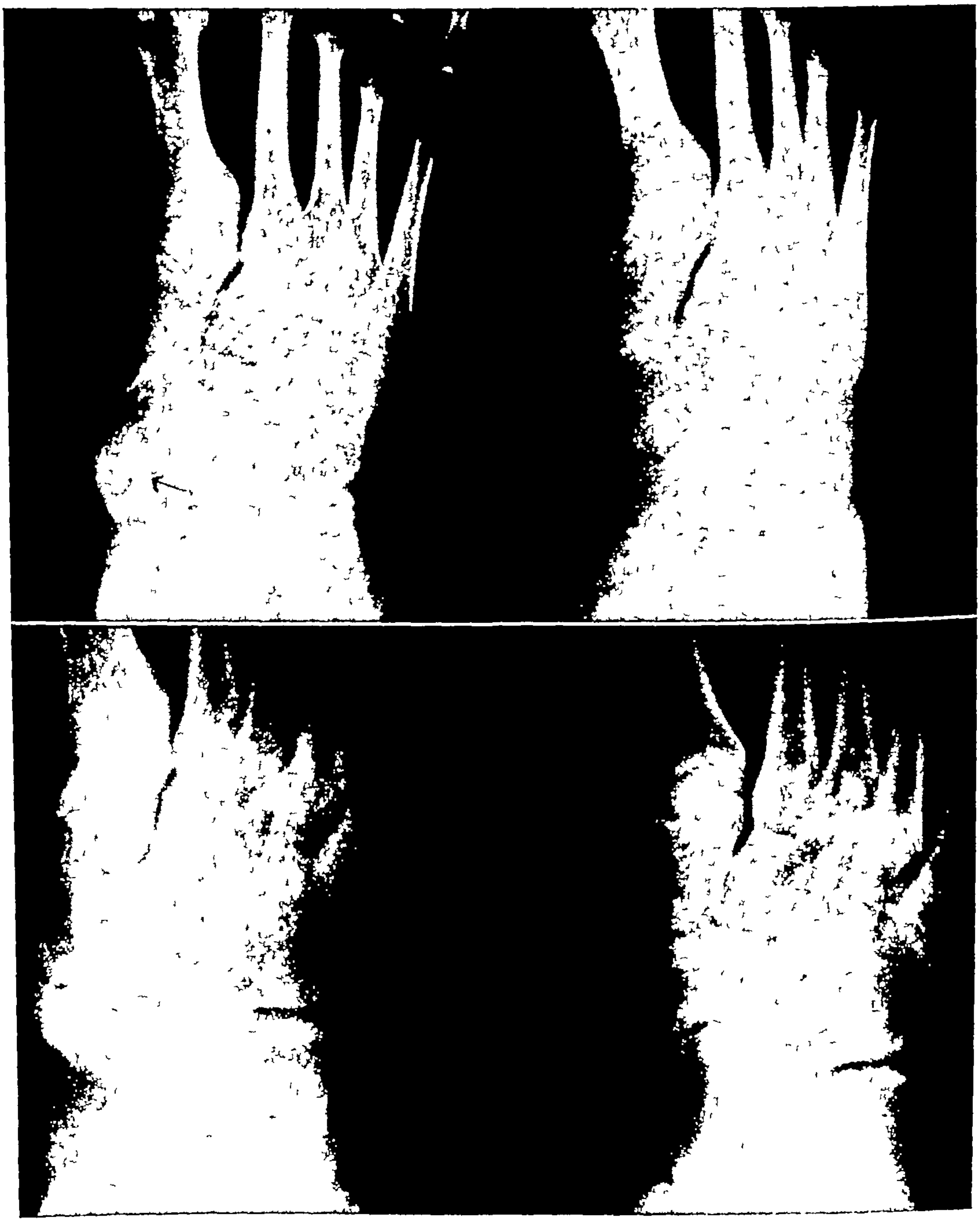
Bifurcated Medial Cuneiform

Horizontal bifurcation of the medial cuneiform is rare I am not aware of its mention in publications Fig 377 shows an asymptomatic case Fig 378 shows an asymptomatic anomalous medial cuneiform

Polydactylism

Accessory toes are frequent Polydactylism often becomes a major handicap Aside from cosmetic offensiveness, the problem varies from discomfort as a result of crowding the shoe to complete inability to wear a standard shoe. The variations in type of polydactylism are numerous, the commonest being a *sixth*

Fig 375



A.

B.

Fig 376

Figs 375 and 376 —*A*, Preoperative accessory navicular *B*, Same foot postoperatively

Fig 379

A B

A. B

Fig 380

Fig 379 —*A*, Polydactylism with extra phalanges incorporated into one toe *B*, Same case, postoperatively

Fig 380 —*A*, Polydactylism, completely separate toes *B*, Same case, postoperatively

Fig 377.

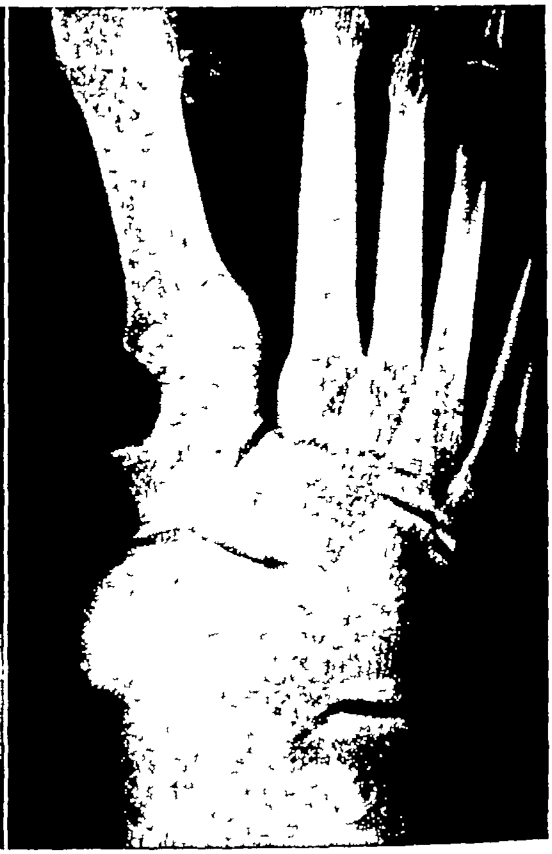


Fig 378

Fig 377 —Horizontal bifurcation of medial cuneiform
Fig 378 —Anomalous medial cuneiform, asymptomatic

toe or an additional hallux *Syndactylism* is usually associated with the anomaly The extra phalanges may be incorporated in one toe (Fig 379) or appear as completely separate toes (Fig 380), often they are accompanied by accessory metatarsals which are fused with the adjacent metatarsal at the base (Figs 381 and 382), or the metatarsal bone may assume a bizarre shape (Fig 383) Hereditary tendency seems apparent In most of my cases, others in the immediate family or ancestry had a similar deformity (Fig 384) Hurwitz encountered an accessory hallux in a little girl whose mother and maternal grandmother had a similar anomaly (Fig 385)

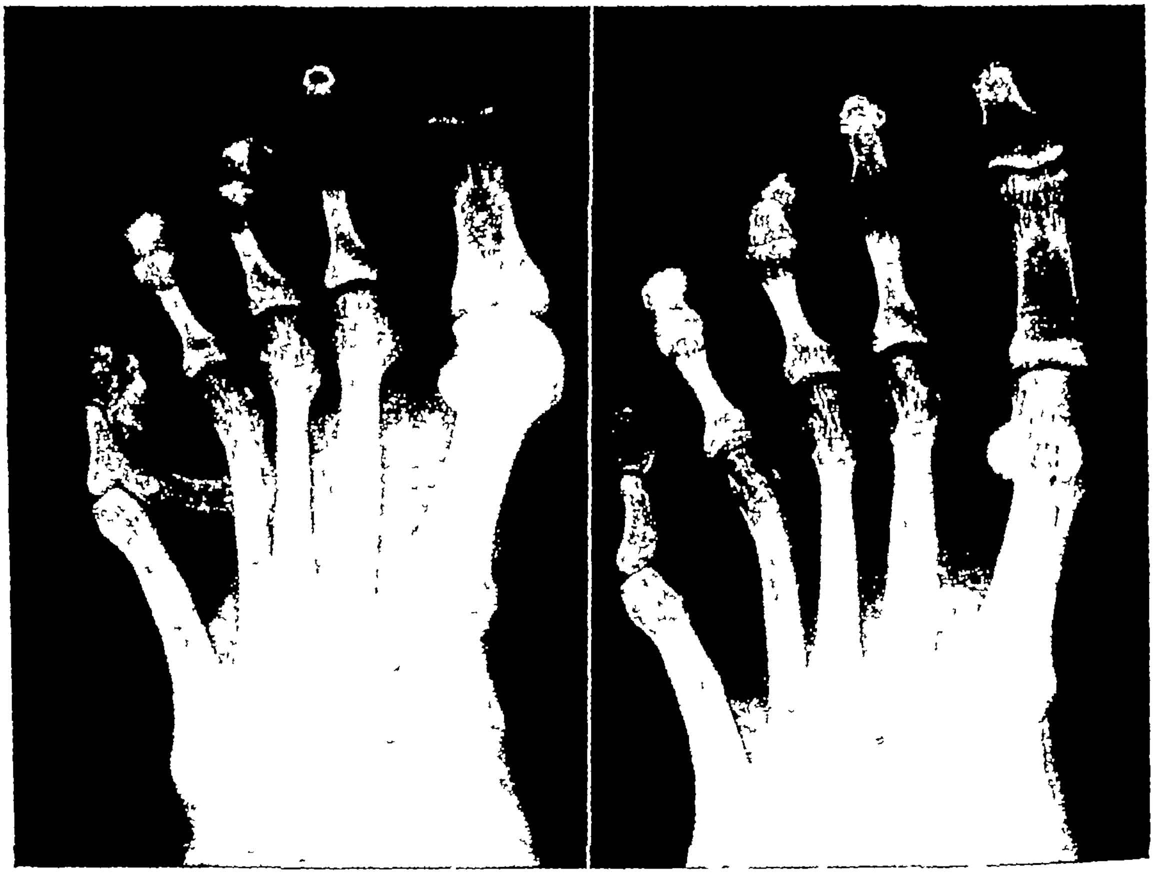
Treatment.—Extreme deformity is an operative problem The technique chosen must meet the requirement of the presenting case, the selection of the structures to be removed must be made with the idea of providing normal contour to the foot as well as of retaining normal muscle function Treatment is usually sought during childhood, because serious cases are self-evident from infancy It is therefore important to consider the results of the operation on the growth of the foot

The extra lesser toe is usually the same size and shape as the fifth toe and is easily amputated The fifth metatarsal, however, has a wide head to accommodate the proximal phalange of the fifth and sixth toes The fibular side of the

B

C

Fig 382 (cont'd) —For legend see opposite page



A B

Fig 381 —A, Polydactylism, accessory metatarsal lying transversely B, Same case, postoperatively.

A.

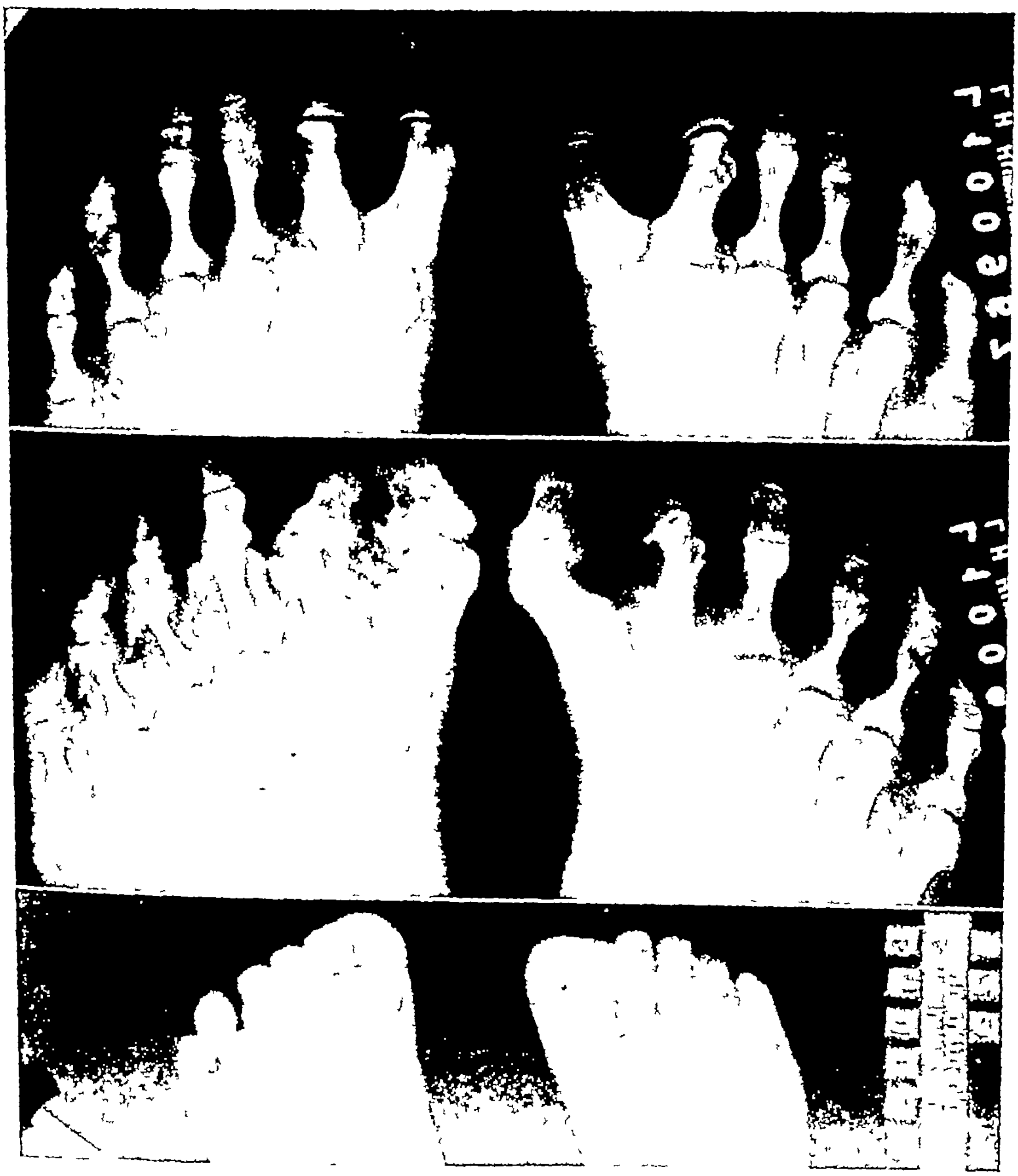
Fig 382 —A, Roentgenogram of bilateral polydactylism, accessory metatarsals fused at base B, Clinical appearance in same case C, Sixteen months postoperatively

head should be amputated, otherwise, it will remain as a projection, subject to constant pressure (Fig 384) The shaft of the fifth metatarsal, moreover, is also wider in these cases than normal, therefore, the entire fibular side of this metatarsal must be amputated The operative technique is as follows

1 Make a longitudinal incision, beginning in the fifth web, extending it proximally and lateralward and ending anterior to the base of the fifth metatarsal.

2. Begin a second incision, also in the web, coursing plantarwise around the base of the sixth toe and continuing on the lateral side of the metatarsal to meet the proximal end of the first incision Remove the extra toe and its appendages, then amputate the excess metatarsal.

3 Suture the fascia and skin in layers. Ambulation may begin in three days.



A

B

C

Fig 385—Accessory hallux and syndactylism in three generations *A*, Mother's *B*, Grandmother's *C*, Child's (Courtesy Dr. Lester H Hurwitz)

Fig 383 —Bizarre accessory metatarsal

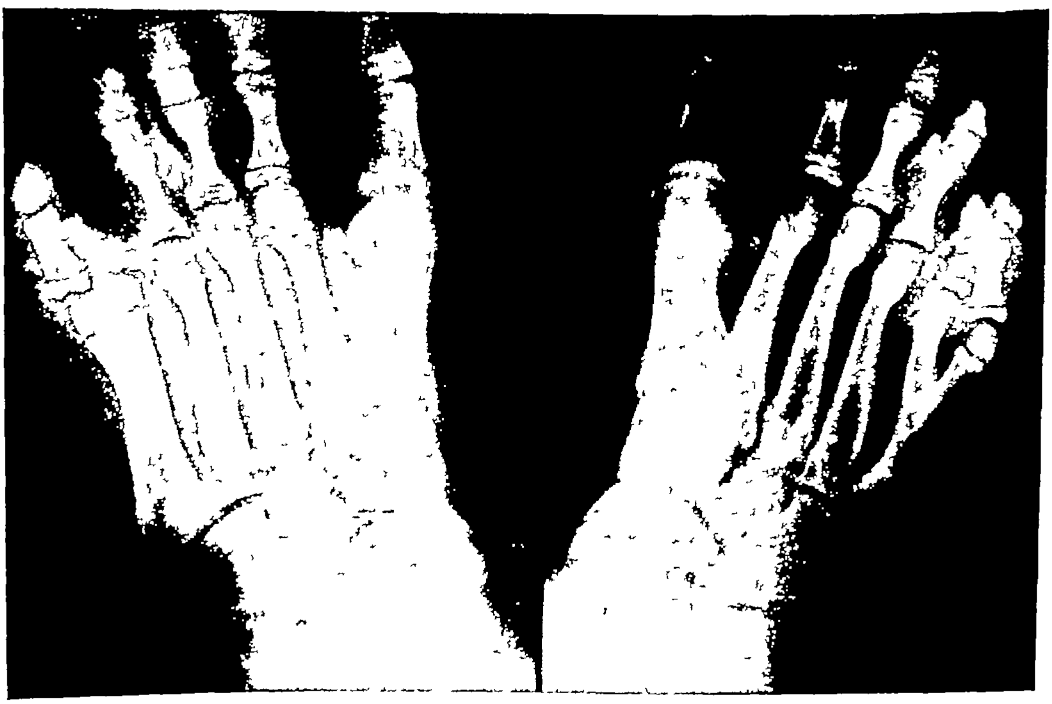


Fig 384 —Roentgenogram of foot of brother of patient represented in Fig 382, A Note fifth digits amputated in infancy without reshaping of metatarsals Remaining epiphysis of base of proximal phalanges and accessory metatarsals became constantly pressured points

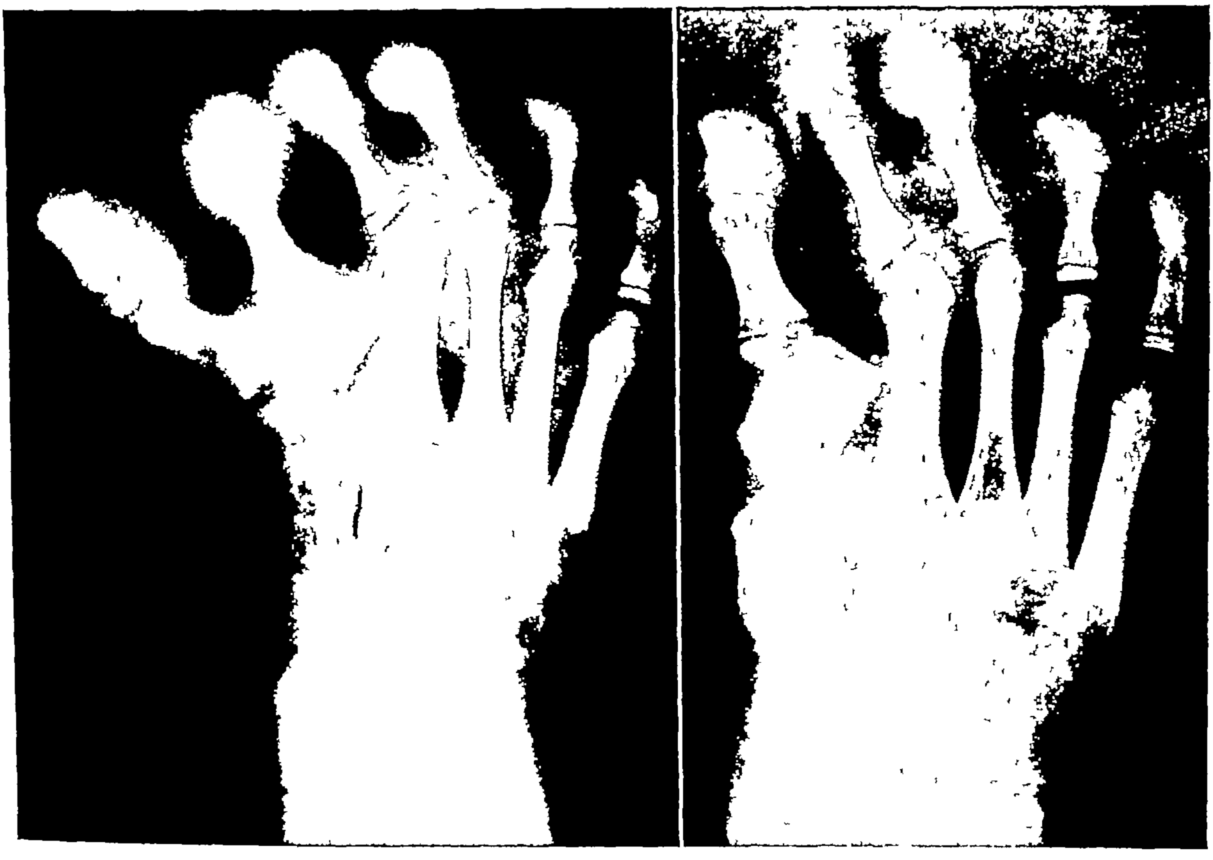
Syndactylism

Webbed toes hardly ever become symptomatic. Treatment is sought for cosmetic reasons

1 Make a horizontal incision over the dorsal margin of one of the webbed toes Denude the skin to the dorsum of the adjacent toe.

2 Make a similar incision on the plantar surface of the adjacent toe and denude the skin to the edge of the toe first incised.

3 Use the dorsal skin flap to cover the denuded surface of one toe and suture



A B

Fig 387 —*A*, Bizarre type of accessory hallux of first metatarsal *B*, Same case, postoperatively

4 Use the plantar skin flap to cover the denuded surface of the adjacent toe and suture

5 Keep the toes separated with paraffin mesh, Telfa dressings, or petrolatum gauze until healing is complete

Megalodactyly or Macrodactylism

Local gigantism of one or more toes is sometimes encountered (Fig 389) The condition may interfere with wearing normal shoes, it may be unsightly Partial amputation of the toe or excision of the middle phalanx or part of the distal half of the proximal phalanx reduces the toe to almost normal size Char-

Sometimes the extra toe is normal, but the fifth toe is abnormal in size and shape If the fifth metatarsal is normal, the fifth toe should be removed through an oval incision at its base, whereas, if the fifth metatarsal bone requires alteration, it is better to remove the sixth normal toe than to reshape the fifth metatarsal on its tibial side (Figs 380 and 382, *C*)

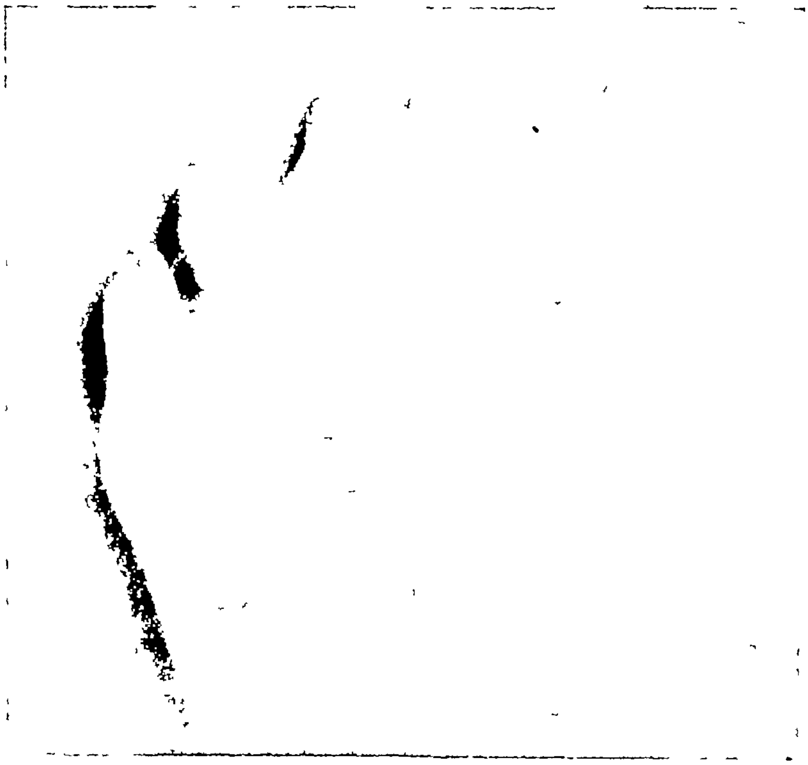
Accessory Hallux

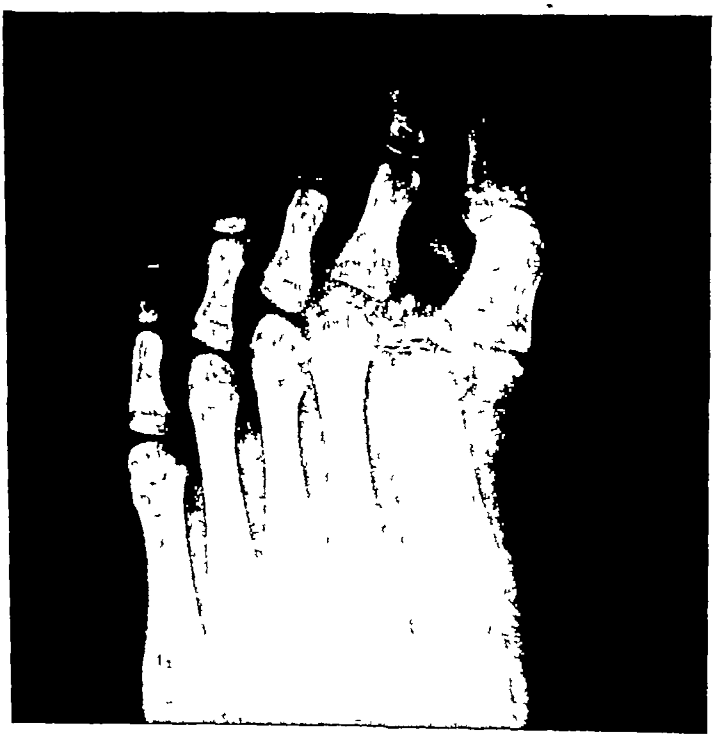
An accessory hallux (Figs 386 and 387) presents a formidable problem because of muscular attachments to the base of the proximal phalanx and

Fig 386 —Accessory distal phalanx of hallux

because of many kinds of deviations The tibialmost toe should always be the one amputated unless the fibularmost toe is grossly deformed or abnormal in size In that case, the abductor hallucis, which is inserted into the base of the proximal phalanx of the fibularmost hallux, must be sutured to the proximal phalanx of the remaining hallux, otherwise, in a growing child the remaining abductor hallucis will gradually pull the toe into a serious hallux varus (Fig 388)

C

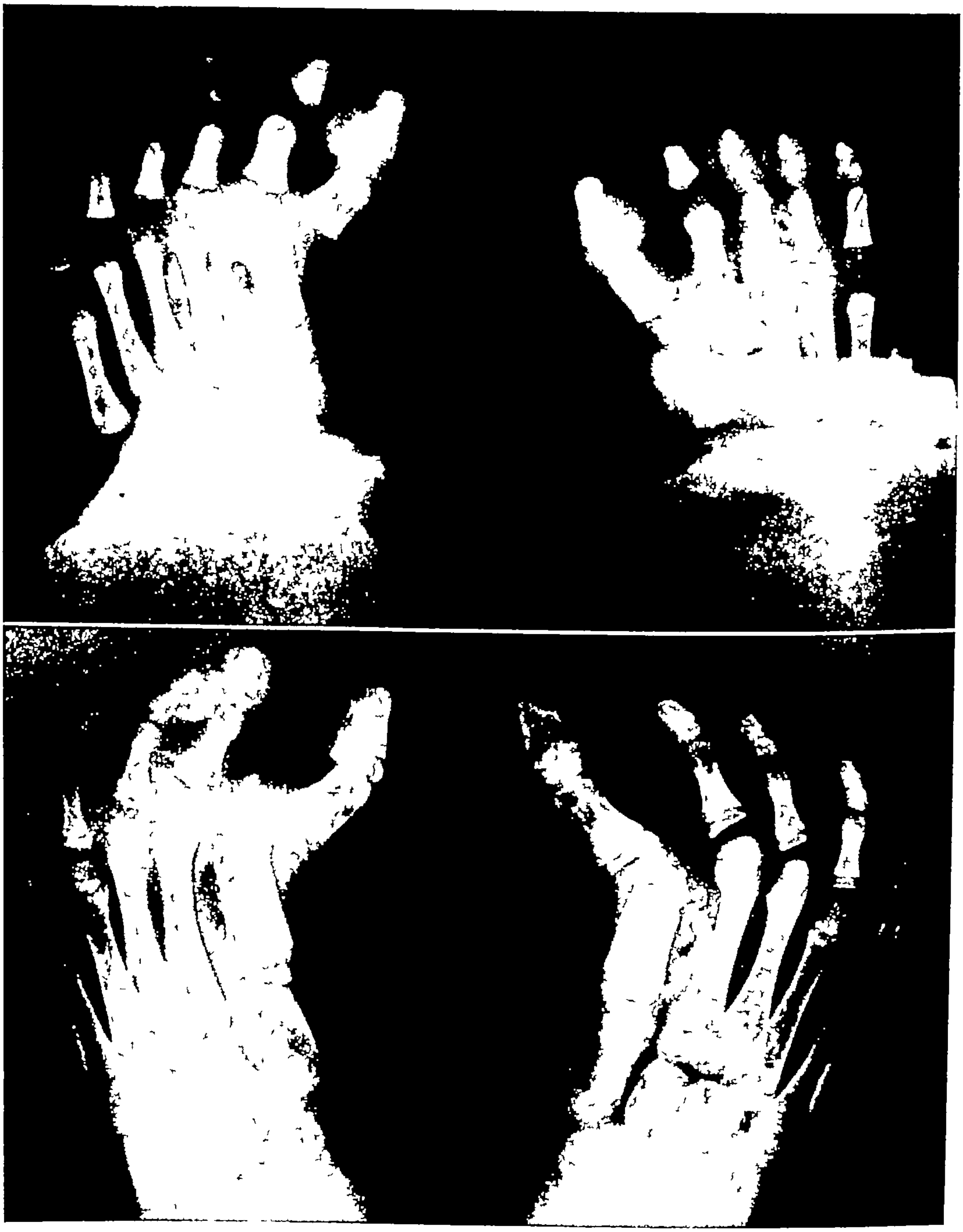




D

Fig 388 (cont'd) —For legend see opposite page

A



B

Fig 388—*A*, Infant with bilateral accessory hallux During infancy, both great toes on fibular side of accessory hallux were amputated (elsewhere) *B*, Same patient at age 6 years Note extreme hallux varus, caused by loss of adductor hallucis insertion *C*, Clinical appearance of left foot shown roentgenographically in *B* Note extreme contraction of abductor hallucis and skin over tibial side of first metatarsophalangeal joint Extensive repair was required to reduce deformity *D*, Postoperative result.

C

Fig 389 (cont'd) —For legend see opposite page

ters (1957) presented an excellent discussion in conjunction with a report of a case regarding the possible relation between neurofibromatosis and macrodactylism as well as other etiologic theories

CONGENITAL CLEFT FOOT AND ABSENCE OF PART OF FOOT

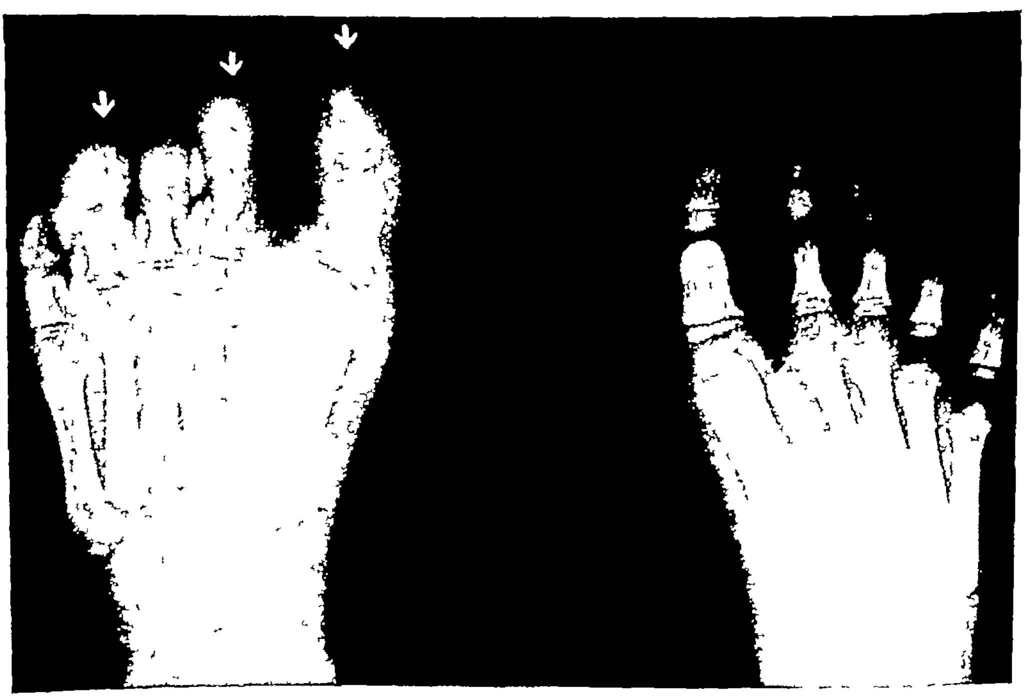
Congenital cleft foot is a troublesome deformity which varies in type and degree Usually one or more toes and some or all of the metatarsal bones are absent, often part of the tarsus is also missing The first and fifth rays are often present (Figs 390 and 391)

Treatment must be directed toward improving function and only secondarily toward cosmetic improvement Bony prominences which become pressured areas should be excised All superfluous tissue that interferes with function should also be removed

HIGH DORSUM OF BASE OF FIRST METATARSAL-FIRST CUNEIFORM JOINT

Congenital enlargement of the dorsum of the first metatarsal-first cuneiform joint is common (Fig 392) It becomes symptomatic only from friction and pressure of the shoe, usually in early adult life and mostly in women Some of the resulting problems are as follows (1) Acute cellulitis over the dorsum of the joint, which may or may not extend down to the bone, producing osteomyelitis The infection enters through a hair follicle in the skin over the bony prominence (2) Tenosynovitis of the extensor hallucis longus tendon, which

A.



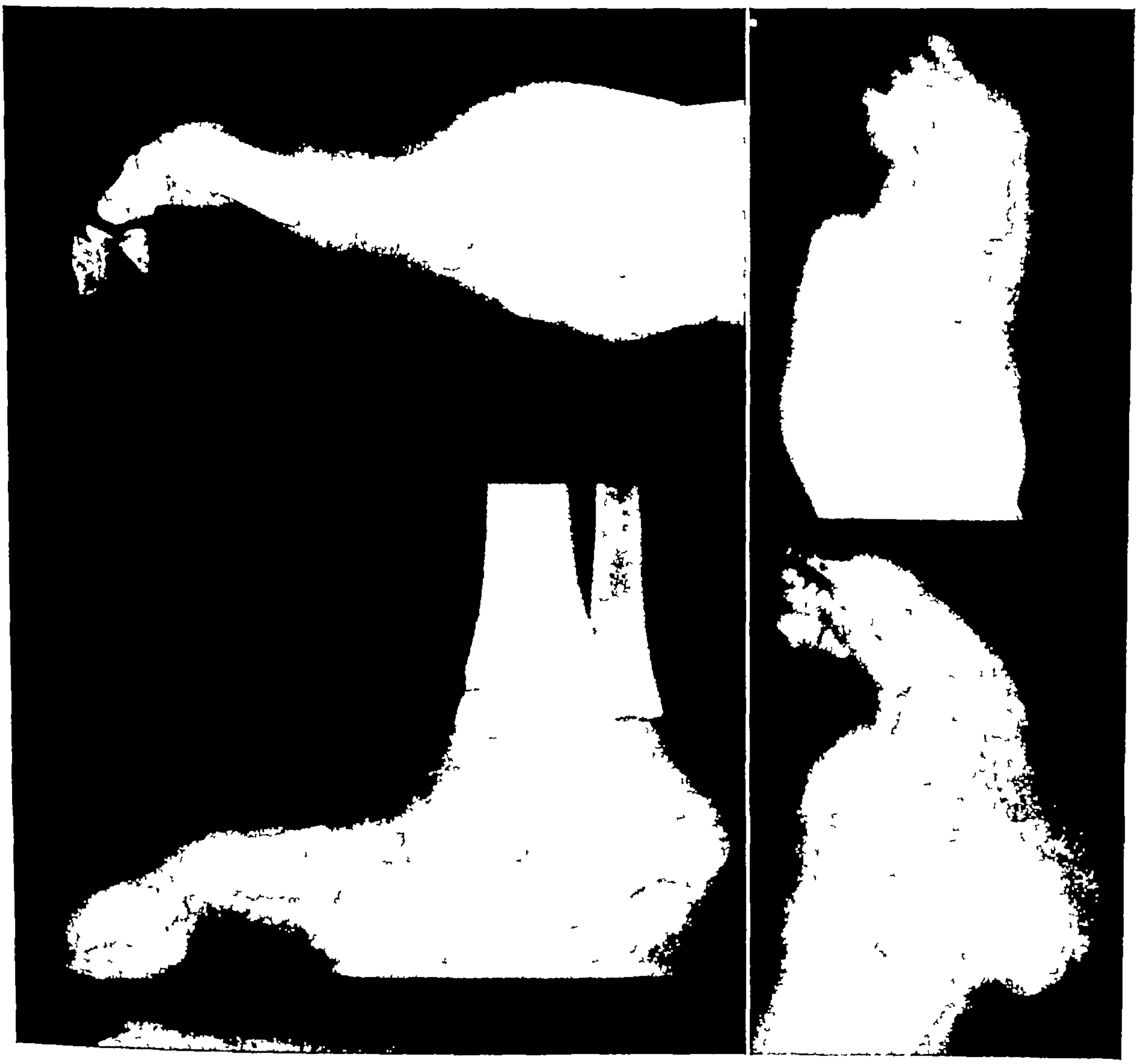
B

Fig 389 —*A*, Megalodactyly of second and third toes *B*, Megalodactyly of first, second, and fourth toes of left foot. *C*, Megalodactyly of second toe of left foot (seen at right in illustration)

C.

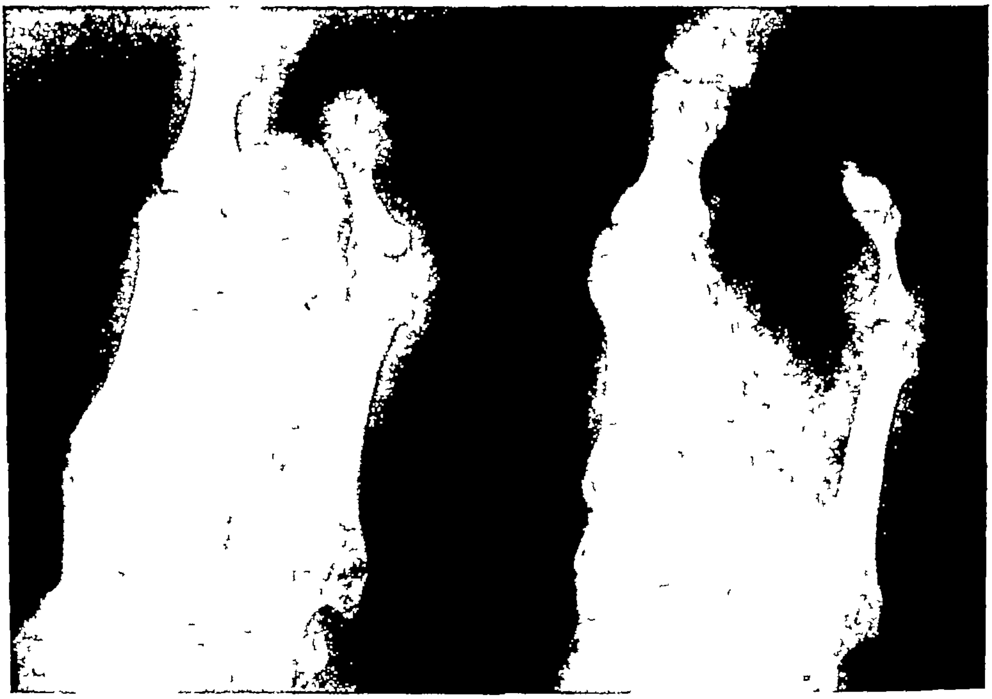
Fig 390 (cont'd) —For legend see opposite page

A



B

Fig 391 —*A*, Congenital absence of most of foot Only first cuneiform-first metatarsal and hallux present *B*, Congenitally bizarre foot



A.

B

Fig 390 —A and B, Congenital cleft foot C, Clinical appearance of case shown in B

should leave a moderate depression, which allows for some new bone formation that may result from surgical irritation. Place additional dressing material over the depressed area to prevent subdermal bleeding into a pocket.

4 Close the fascia and skin in layers and apply a compression bandage.

LIPPING ON DORSUM OF TALONAVICULAR JOINT

Lipping at the talonavicular joint is not uncommon Because the hypertrophied joint margins collide when the foot is dorsiflexed, the condition is painful (Fig 393) It may or may not be associated with rigid or semirigid flatfoot The lipping is always more pronounced on the dorsum of the head of the talus than on the navicular

Etiology.—The etiology is obscure Probably a congenital defect of the joint occurs in which the surfaces are flat where the talus and navicular meet at the dorsum. Normally this joint has a ball-and-socket relationship, whereas if the surfaces are flat, the two bones collide every time the foot is dorsiflexed for each step The gradual osteophytic changes are a reaction to this trauma. The presumed etiologic mechanism is comparable to that which causes hallux rigidus

Symptoms.—Lipping is usually unilateral, when it is bilateral, only one side ordinarily produces symptoms. The condition may be symptomless and disclosed only during routine roentgenologic examination. The symptomatic cases begin in middle life and oftener in women.

The disability is a mildly or severely painful swelling over the talonavicular joint The patient cannot bear weight because of pain, which is aggravated when the foot is dorsiflexed

Routine roentgenographic views of the talonavicular joint verify the diagnosis.

Recommended Treatment.—Symptoms respond to arthroplasty of the joint by the following procedure

1 Make a semilunar incision over the dorsum of the body of the navicular, following the transverse semielliptical course of this bone, and retract the skin margins

2 Incise the ligaments and capsule somewhat proximally to the skin incision, underscoring and retracting its margins and exposing the lipping of the talonavicular joint

3 Remove the excess bone with an osteotome

4 Manually dorsiflex the foot to make certain that the dorsal margins of the talonavicular joint have ceased to collide

5 The rounding off of the head of the talus should be accentuated to allow for some new growth of bone which may result from surgical irritation (Fig 394)

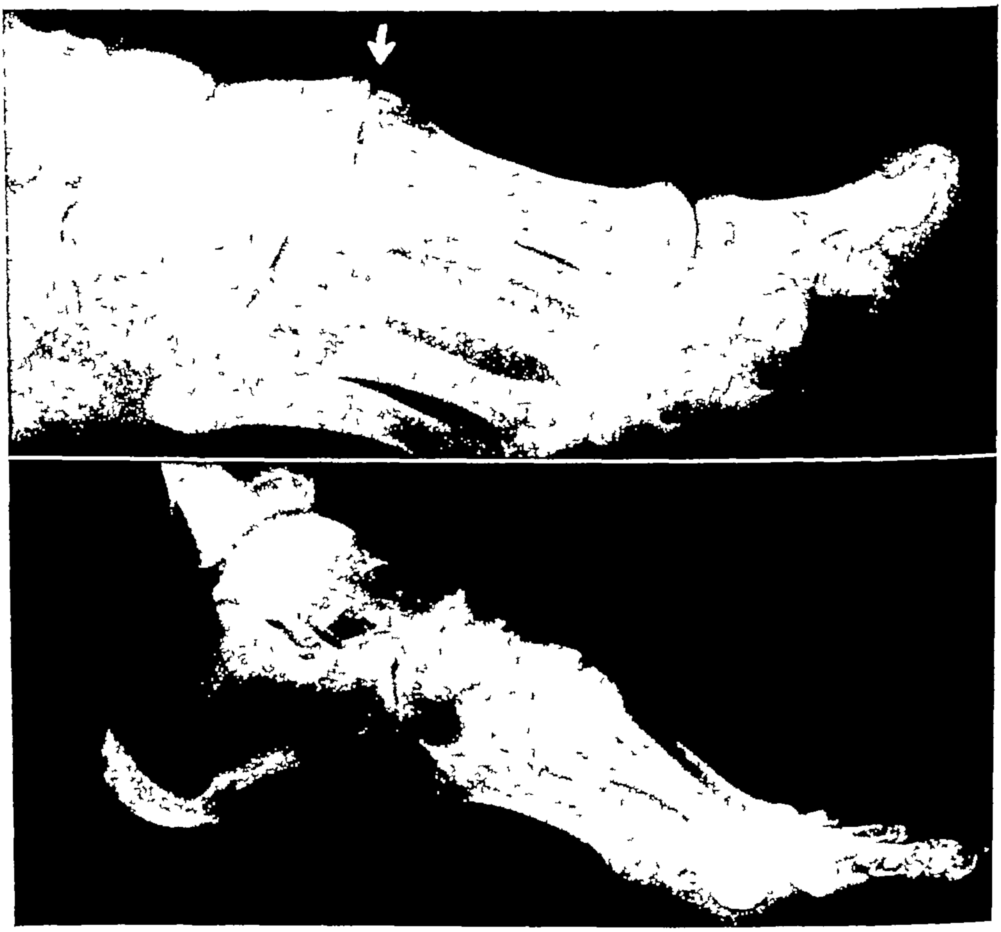
6 The capsule and ligament may now be closed with fine chromic catgut, after 10 mg of hydrocortisone have been instilled to reduce postoperative pain and scar formation

7 Suture the skin and apply a compression bandage for from twelve to twenty-four hours Ambulation may begin in forty-eight hours. The area usually heals uneventfully In about six weeks the patient is completely free of pain.

lies immediately over this enlargement. (3) Constant friction and pressure over the area gradually producing a fibrotic mass, which may break down and form a chronic draining sinus (4) Osteophytic changes in the form of lipping at the base of the first metatarsal and medial cuneiform

Treatment.—In moderate cases, avoiding shoes that exert pressure and friction over the area ameliorates symptoms In protracted cases, especially when lipping has formed, removal of the bony prominence is indicated

A.



B

Fig 392 —*A*, High dorsum of base of first metatarsal-first cuneiform articulation *B*, Postoperative appearance

The following technique makes ambulation possible in forty-eight hours Healing is usually uneventful and complete in from six to eight weeks

1 Make a longitudinal incision along the dorsomedial aspect of the base of the first metatarsal-first cuneiform

2 Retract the lateral skin margin and make a longitudinal incision in the ligaments and capsule over the middle of the joint This will obviate having two lines of sutures in the same plane

3 Retract the fascial margins to expose the bony prominence, which is removed with an osteotome Smooth with a rasp Removal of the bony prominence

should leave a moderate depression, which allows for some new bone formation that may result from surgical irritation. Place additional dressing material over the depressed area to prevent subdermal bleeding into a pocket.

4. Close the fascia and skin in layers and apply a compression bandage.

LIPPING ON DORSUM OF TALONAVICULAR JOINT

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Etiology.—The etiology is obscure. Probably a congenital defect of the joint occurs in which the surfaces are flat where the talus and navicular meet at the dorsum. Normally this joint has a ball-and-socket relationship, whereas if the surfaces are flat, the two bones collide every time the foot is dorsiflexed for each step. The gradual osteophytic changes are a reaction to this trauma. The presumed etiologic mechanism is comparable to that which causes hallux rigidus.

Symptoms.—Lipping is usually unilateral; when it is bilateral, only one side ordinarily produces symptoms. The condition may be symptomless and disclosed only during routine roentgenologic examination. The symptomatic cases begin in middle life and oftener in women.

The disability is a mildly or severely painful swelling over the talonavicular joint. The patient cannot bear weight because of pain, which is aggravated when the foot is dorsiflexed.

Routine roentgenographic views of the talonavicular joint verify the diagnosis.

Recommended Treatment.—Symptoms respond to arthroplasty of the joint by the following procedure:

1. Make a semilunar incision over the dorsum of the body of the navicular, following the transverse semielliptical course of this bone, and retract the skin margins.

2. Incise the ligaments and capsule somewhat proximally to the skin incision, underscoring and retracting its margins and exposing the lipping of the talonavicular joint.

3. Remove the excess bone with an osteotome.

4. Manually dorsiflex the foot to make certain that the dorsal margins of the talonavicular joint have ceased to collide.

5. The rounding off of the head of the talus should be accentuated to allow for some new growth of bone which may result from surgical irritation (Fig. 394).

6. The capsule and ligament may now be closed with fine chromic catgut, after 10 mg. of hydrocortisone have been instilled to reduce postoperative pain and scar formation.

7. Suture the skin and apply a compression bandage for from twelve to twenty-four hours. Ambulation may begin in forty-eight hours. The area usually heals uneventfully. In about six weeks the patient is completely free of pain.

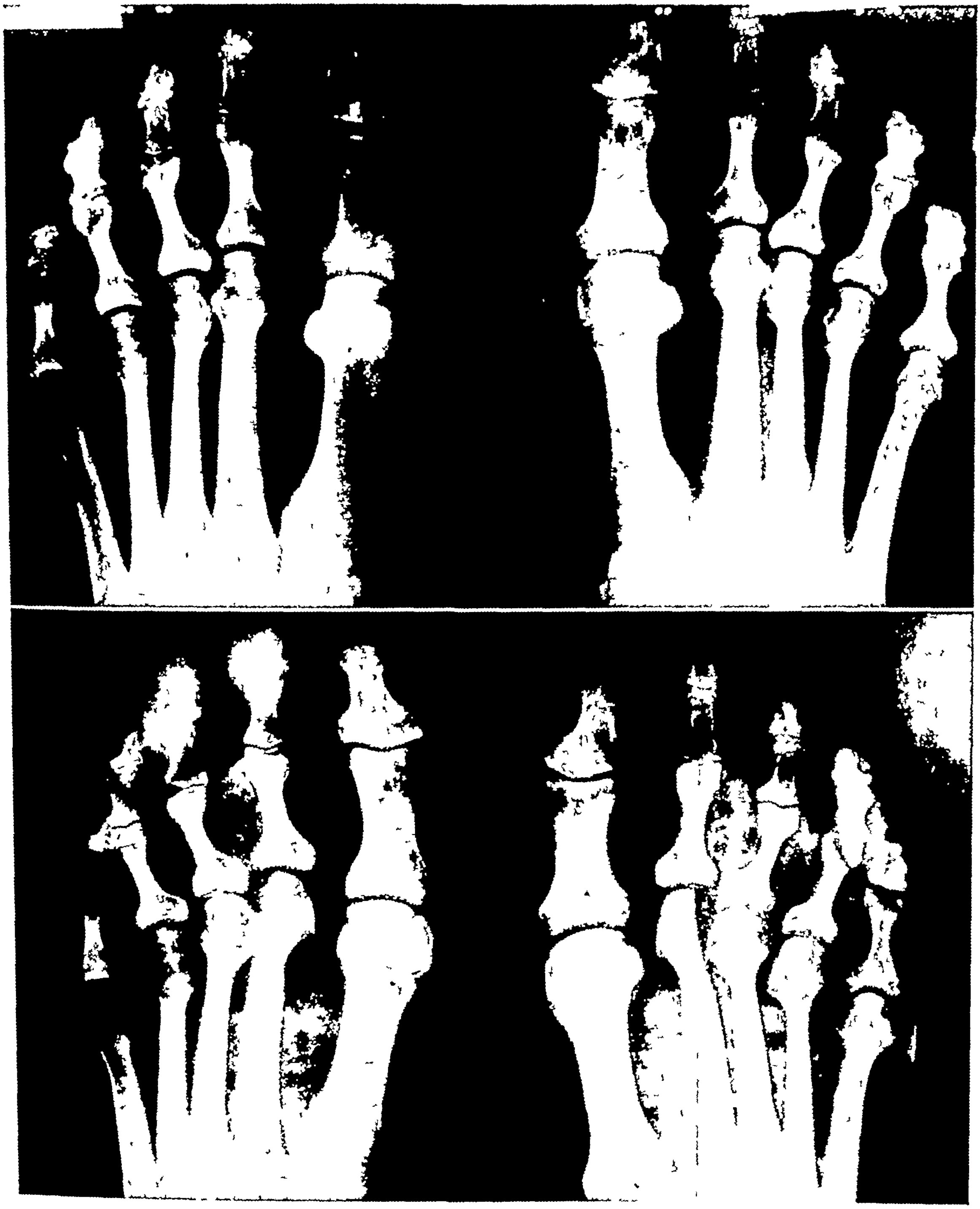
Fig 393

Fig 394

Fig 393 —Lipping on dorsum of talonavicular joint, pain on dorsiflexion

Fig 394 —Same case as shown in Fig 393, after removal of lipping

A



B

Fig 395 — *A*, Metatarsus proximus between second and third metatarsal heads *B*, Same case, postoperatively

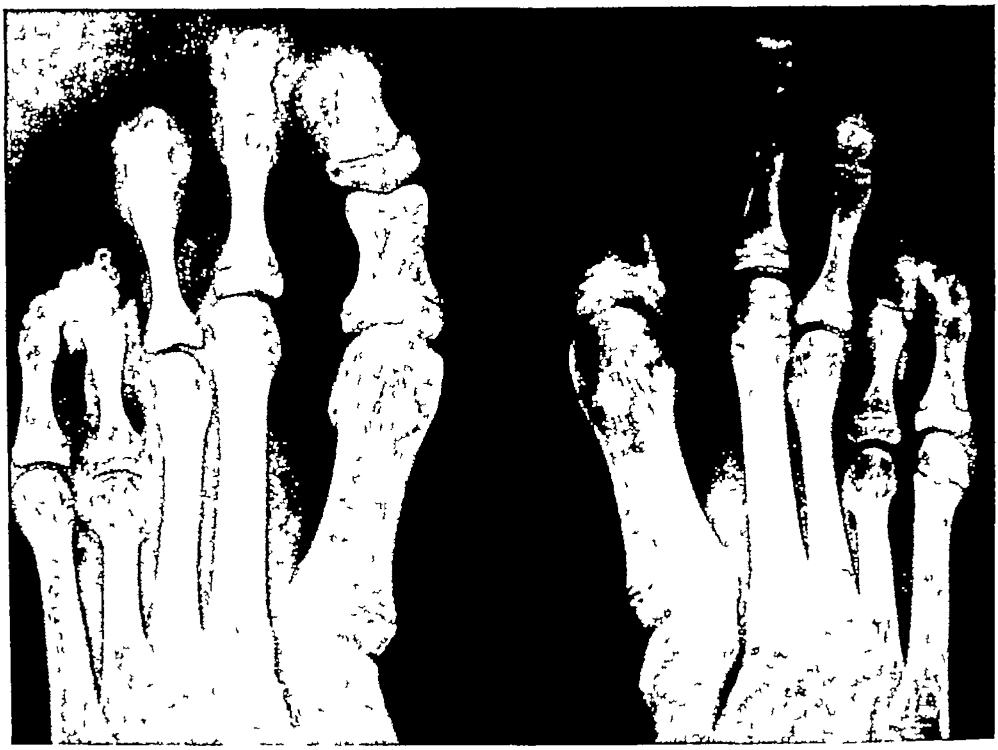


Fig 396 —Bilateral congenital short fourth metatarsal.

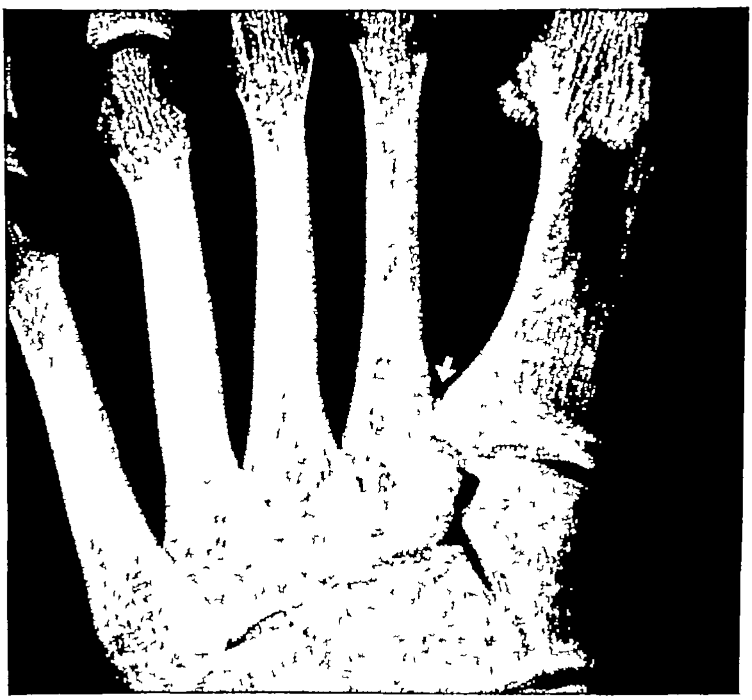


Fig 397 —Base of first metatarsal grates into base of second metatarsal Note aseptic necrosis in base of second metatarsal

Fig 398

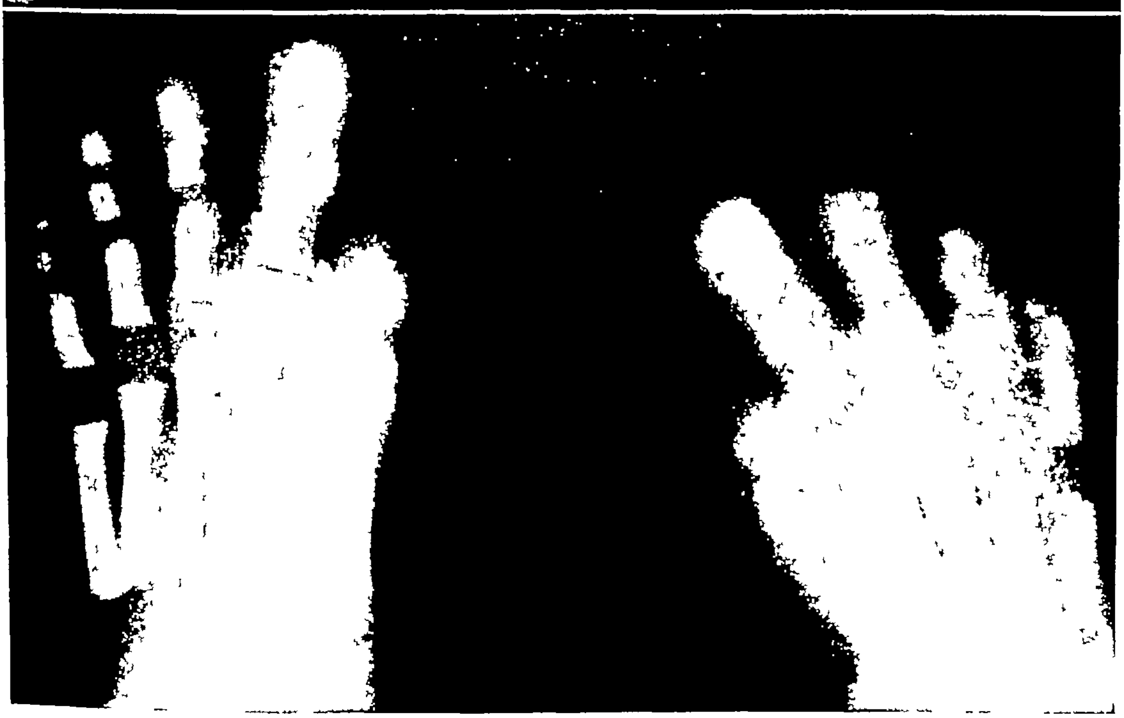


Fig 399

Fig 398 –Congenital deformity of talonavicular joint causes unstable foot

Fig 399 –Bilateral congenital underdevelopment of first metatarsal

METATARSUS PROXIMUS

Occasionally two metatarsal heads are so close that they rub against each other, causing pain from friction and requiring excision of the side of one of the heads (Fig 395).

CONGENITAL SHORT FOURTH METATARSAL

Congenital short fourth metatarsal is common (Fig 396) At times the condition becomes symptomatic

Fig 400 —Congenital anomaly of posterior tuberosity of calcaneus Painful from pressure of shoes

SOME RARE ANOMALIES

Some rare anomalies that may become symptomatic are illustrated in Figs 397 to 400, which show, respectively, (1) aseptic necrosis in the base of the second metatarsal as a result of grating of the base of the first metatarsal into the second, (2) anomaly of posterior tuberosity of calcaneus which became painful from pressure of shoes, (3) unstable foot caused by deformity of the talonavicular joint, (4) bilateral underdevelopment of first metatarsal

REFERENCES

Austin, F H Symphalangism and Related Fusing of Tarsal Bones, J Internat Coll Surg 56 882-885, June, 1951
Bechtol, C O, and Mossman, H W Club-Foot, An Embryological Study of Associated Muscle Abnormalities, J Bone & Joint Surg 32A:827-836, Oct, 1950
Bizarro, A H On Sesamoid and Supernumerary Bones of the Limbs, J Anat. 55 256, 1921 (Cited by McDougall)
Bjornson, R G B Developmental Anomaly of the Lateral Malleolus Simulating Fracture, J Bone & Joint Surg 38A 128-130, Jan, 1956

Blumenfeld, I, Kaplan, N, and Hicks, E O. The Conservative Treatment of Congenital Talipes Equinovarus, J Bone & Joint Surg 28 765-786, Oct, 1946

Browne, Denis Congenital Malformations, Practitioner 131 20, July, 1933

Charters, A D Local Gigantism, J Bone & Joint Surg 39B 542-547, Aug, 1957

Chollett, B G Relapsed or Resistant Clubfoot of Early Childhood, J A M A 101 1866-1869, Dec 9, 1933

Davis, L A, and Hatt, W. S Congenital Abnormalities of the Feet, Radiology 64 818-825, June, 1955

Farmer, A W Congenital Elephantiasis Associated With Constriction by Anomalous Bands, J Bone & Joint Surg 30B·606-612, Nov, 1948

Garceau, G J, and Manning, K R Transposition of the Anterior Tibial Tendon in the Treatment of Recurrent Congenital Clubfoot, J Bone & Joint Surg 29 1044-1047, Oct, 1947

Geist, E S Accessory Scaphoid Bone, J Bone & Joint Surg 7 570, July, 1925

Harris, R I, and Beath, T Etiology of Peroneal Spastic Flatfoot, J Bone & Joint Surg 30B 624, 1948

Harris, R I, and Beath, T John Hunter's Specimen of Talocalcaneal Bridge, J Bone & Joint Surg 32B 203, May, 1950

Harris, R I Rigid Valgus Foot Due to Talocalcaneal Bridge, J Bone & Joint Surg 37A 169-183, Jan, 1955

Hayden, W de G, and Murray, R A Pedal Anomaly, A M. A Arch Surg 72 421-425, March, 1956

Hurwitz, L H Personal communication

Jack, E A Bone Anomalies of the Tarsus, in Relation to Peroneal Spastic Flatfoot, J Bone & Joint Surg 36B 530-542, Nov, 1954

Kidner, F C Prehallux (Accessory Scaphoid) in Its Relation to Flat-Foot, J Bone & Joint Surg 11 831, Oct, 1929

Kidner, F C Prehallux in Relation to Flatfoot, J A M A 101 1539-1542, Nov 11, 1933

Kite, J H The Treatment of Congenital Clubfeet, Study of Results in 200 Cases, J A M A 99 1156-1162, Oct 1, 1932

Kite, J H Principles Involved in the Treatment of Congenital Club-Foot, J Bone & Joint Surg 21 595-606, July, 1939

Kite, J H Treatment of Congenital Clubfeet and of Congenital Metatarsus Varus, Twenty-Fourth Annual Meeting, American Academy of Orthopaedic Surgeons, Chicago, Jan 29, 1957

Knight, R A Developmental Deformities of the Lower Extremities, J Bone & Joint Surg 36A 521-527, June, 1954.

Lloyd-Roberts, G C, and Spence, A J Congenital Vertical Talus, J Bone & Joint Surg 40B 33-41, Feb, 1958

de Marchi, E, Gambier, R, and Vespignani, L Les synostoses tarsiennes dans le pied plat valgus douloureux, J radiol et électrol 36 665-674, 1955

Morrison, A B Os Paracuneiforme, Some Observations on an Example Removed at Operation, J Bone & Joint Surg 35B 254-255, May, 1953

McDougall, A Os Trigonum, J Bone & Joint Surg 37B 257-265, May, 1955

O'Rahilly, Ronan A Survey of Carpal and Tarsal Anomalies, J Bone & Joint Surg 35A 626-642, July, 1953

Osmond-Clarke, H Congenital Vertical Talus, J Bone & Joint Surg 38B 334-341, Feb, 1956

Pfitzner, W Beitrage zur Kenntnis des menschlichen Extremitatenskelets VII Die Variationen im Aufbau des Fusskelets, In Schwalbe, G Morphologischen Arbeiten, Jena, 1896, Gustav Fischer, vol, 6, p 235

Sante, L R Principles of Roentgenological Interpretation, ed 5, Ann Arbor, Mich, 1944, Edwards Brothers, Inc, pp 154-155

Scherb, R Zur Aetiologie kongenitaler und kongenitalbedingter Fussdeformitaten mit besonderer Berucksichtigung des Pes equino-varus congenitus, Acta chir scandinav 67 717-750, 1930

Shands, A R, Jr, and Wentz, I J Symposium on Orthopedic Surgery, Congenital Anomalies, Accessory Bones, and Osteochondritis in the Feet of 850 Children, S Clin North America 33 1643-1666, 1953

Slomann, H C On Coalitio Calcaneo-Navicularis, J Orthop Surg 3.586, Nov, 1921

Stewart, S F Club-Foot Its Incidence, Cause and Treatment, An Anatomical-Physiological Study, J Bone & Joint Surg 33A 577-590, July, 1951

Thomson, S A Treatment of Congenital Talipes Equinovarus With a Modification of the Denis Browne Method and Splint, J Bone & Joint Surg 24 291, April, 1942

Thomson, S A Treatment of Congenital Clubfoot, J Bone & Joint Surg 31A 431-434, April, 1949

Thomson, S A Modified Denis Browne Splint for Unilateral Club-Foot to Protect the Normal Foot, J Bone & Joint Surg 37A 1286, Dec, 1955

Trevor, David Tarso-Epiphysial Aclasis, J Bone & Joint Surg **32B** 204-211, May, 1950
Vaughan, W H, and Segal, G Tarsal Coalition With Special Reference to Roentgenographic Interpretation, Radiology **60** 855-863, June, 1953
Waugh, W Partial Cubo-Navicular Coalition as a Cause of Peroneal Spastic Flat Foot, J Bone & Joint Surg **39B** 520-523, Aug, 1957
Webster, F S, and Roberts, W M Tarsal Anomalies and Peroneal Spastic Flatfoot, J A M A **146** 1099-1104, July 21, 1951
White, J W, and Gulledge, W H Skin-Tight Casts for Treatment of Club-Foot, J Bone & Joint Surg **33A** 475-498, April, 1951
Zadek, I Significance of Accessory Tarsal Scaphoid, J Bone & Joint Surg **8** 618-626, July, 1926
Zadek, I, and Gold, A M Accessory Tarsal Scaphoid, J Bone & Joint Surg **30A** 957-968, Oct, 1948

CHAPTER EIGHTEEN

Amputations

IN A STRICT SENSE, AMPUTATION IS THE EXCISION OF A PART OR OF A WHOLE MEMBER through the body of a bone It differs from *disarticulation,* which is excision through a joint Both terms, however, are used interchangeably. In relation to the toes and forefoot, a true amputation is often termed *partial amputation,* and a disarticulation is referred to as *amputation* This distinction is observed in the present discussion Amputations that are discussed herein are mainly those which are performed to remove a deformed, unsightly, or useless part of the foot This differs from the circumstance ordinarily demanding amputation of a limb, namely, the loss of the blood supply of the limb, which makes the extremity not only useless but threatens the life of the patient. Examples of such a circumstance are given only under the topics Amputation of a Metatarsal and Transmetatarsal Amputation because the subject of vascular degeneration leading to amputation is outside the purpose of this textbook.

Prime indications for amputations of the foot or the major parts of a foot are deformities, gangrene due to severe trauma, burns, and vascular occlusive diseases In cases of traumatic gangrene and controlled infection, it is good surgical judgment to allow time for a line of demarcation to form and for collateral circulation to develop before amputation is undertaken Gangrene due to vascular occlusive disease calls for special care in deciding the saw line, because of deficient blood supply proximal to the site of gangrene It cannot be overemphasized that a wound is healed only by the patient's own blood supply

PARTIAL AMPUTATION OF LESSER TOES

Partial amputation of a lesser toe is hardly ever necessary. The only indications for such an operation is complete loss of blood supply or a crushing injury Most deformities of the lesser toes may be corrected by excision of part of the

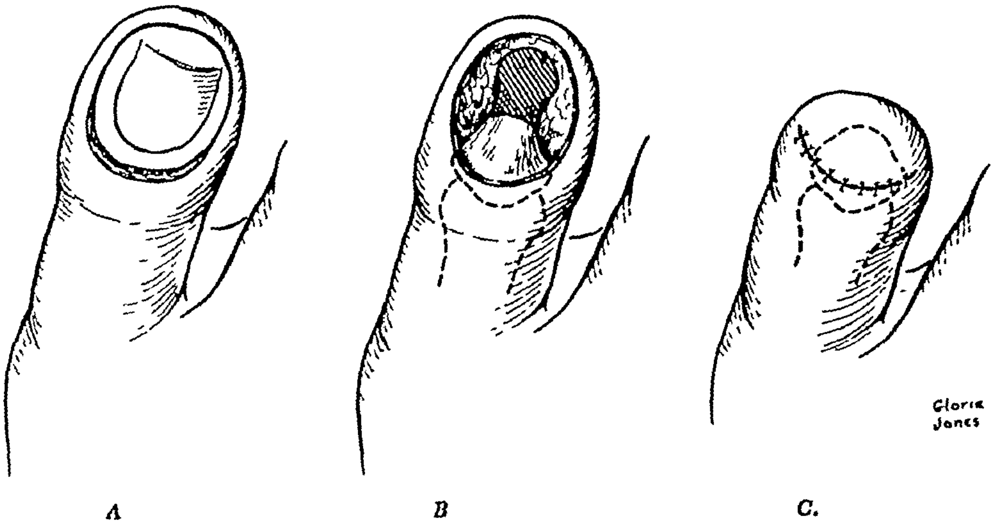


Fig 401.

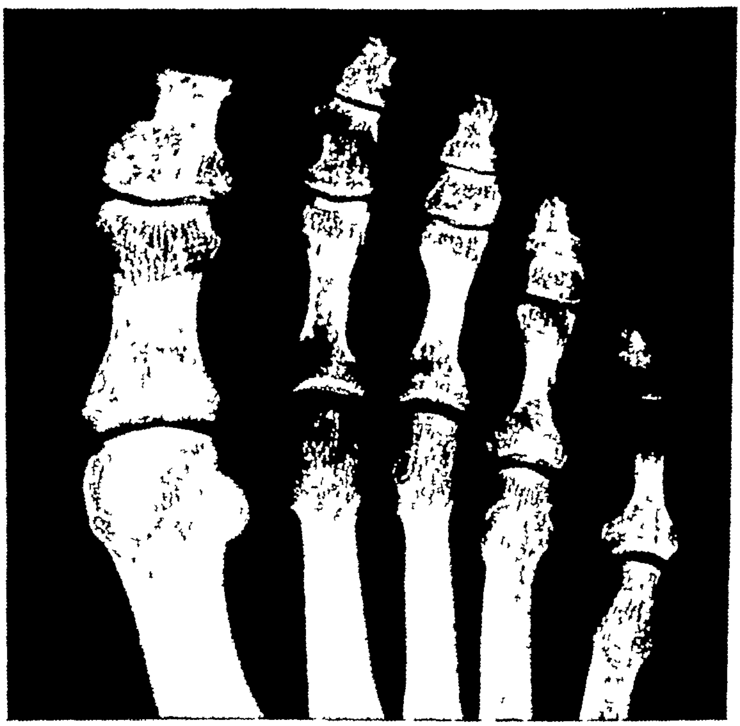


Fig 402

Fig 401 and 402—Partial amputation of distal phalanx of great toe A, Remove skin and nail B, Underscore skin and remove fat. Amputate shaded area C, Skin flap brought over and sutured

middle phalanx, the entire middle phalanx, or the head of the proximal Amputation of the entire proximal phalanx for certain deformities of the toes has been widely practiced. This is an unjustified mutilating operation; the harm done cannot be undone Amputation of the head of the proximal phalanx usually suffices When the base of a proximal phalanx must be amputated, for all practical purposes, the entire toe must be removed, otherwise, the remainder of such a toe becomes a soft unstable appendage which cannot be maintained in normal position

PARTIAL AMPUTATION OF DISTAL PHALANX OF HALLUX (PARTIAL, OR SUBTOTAL, PHALANGECTOMY)

Partial, or subtotal, phalangectomy is indicated for extreme deformity of the nail and distal phalanx of the hallux

1. Make a semilunar incision, starting on either side, about 0 5 cm proximally to the eponychium, over the dorsum of the toe Carry the incision down to the bone and extend it on both sides to about a third of the way to the plantar surface.
2. Begin a second semilunar incision at either end of the initial incision and continue it also completely around the distal end of the toe until it meets both ends of the first incision.
3. Free and remove the entire dorsal flap, including the nail and nail bed, from the dorsum of the distal phalanx.
4. Underscore the remaining intact tissue around the distal phalanx
5. Amputate the distal half of the distal phalanx
6. Round and smooth the stump with a Joseph nasal rasp.
7. The distal flap at this point approximates the initial semilunar incision Suture the margins (Figs 401 and 402) If the distal skin flap must be sutured under tension, the suture line will slough In that case a little more bone should be removed in order that the flap can be sutured without tension It will then heal by first intention

AMPUTATION OF DISTAL PHALANX (COMPLETE, OR TOTAL, PHALANGECTOMY)

Indications for complete, or total, phalangectomy arise mainly in the first and second toes and only occasionally in the three lesser toes When amputation of the distal phalanx of one of the three lesser toes is indicated, it is, in most instances, more practical to amputate the entire toe The second toe, however, must not be amputated except under extreme necessity Once the second toe has been removed, hallux valgus is almost certain to ensue even in a previously normal foot An existing hallux valgus becomes worse, because the hallux moves over into the space left by the amputated second toe

The procedure for amputation of the distal phalanx of the lesser toes is the same as that described for partial amputation of the distal phalanx of the great toe.

DISARTICULATION AT PHALANGEAL JOINT OF GREAT TOE

The indications for disarticulation of the phalangeal joint of the great toe are the presence of osteomyelitis, neoplasms, or crushing injuries of the distal phalanx.

1. Make a semielliptical incision over the dorsum of the hallux, beginning on the side of the head of the proximal and ending at the same point on the opposite side of the phalanx The uppermost curve of the ellipse should course just proximally to the head of the proximal phalanx.

2. Begin a second semielliptical incision at the starting point of the first incision and continue the incision plantarwise and distally to meet at the end of the first incision (Fig 403, *B*).

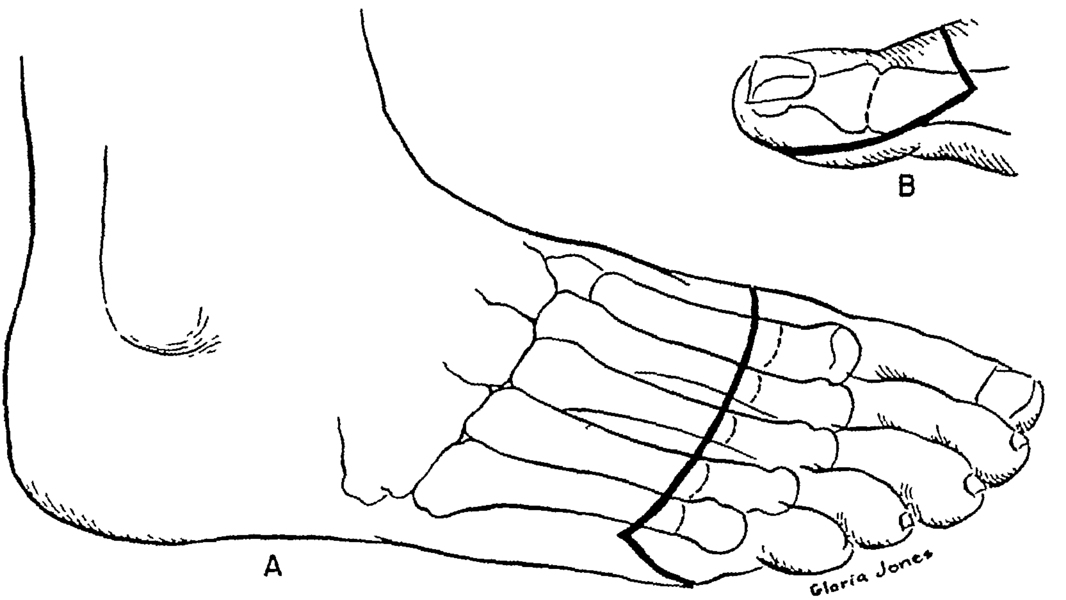


Fig 403 —Amputations, using long plantar flap *A*, Transmetatarsal amputation *B*, Amputation of distal phalanx of great toe

3 Disarticulate the distal phalanx and bring the plantar flap forward to cover the head of the proximal phalanx, suturing the flap to the dorsal margin The plantar flap must always be long enough to permit suturing without tension, otherwise, the suture line sloughs and leaves a wide scar

4 The long flexor and extensor tendons may or may not be sutured to each other, as the operator chooses The sheaths of the tendons adhere to the shaft of the proximal phalanx by circular fibers They retain their function after healing, even when not sutured

DISARTICULATION AT METATARSOPHALANGEAL JOINT OF LESSER TOES

When a lesser toe must be lost, it is best to remove it at the metatarsal joint. Amputation through the proximal phalanx leaves a small useless stump subject to

friction and pressure. Because the stump is so close to the web, it does not look any better than a metatarsophalangeal disarticulation.

1 Make a teardrop incision completely around the base of the toe to be amputated, with the apex of the incision pointing proximally on the dorsum.

2 Plantar-flex the toe, opening the dorsal incision and permitting the metatarsophalangeal joint to be incised on the dorsum and carried around both sides of the joint

3 On dorsiflexion of the toe, section the plantar capsule to permit complete removal of the toe It is not an advantage to suture the flexor and extensor tendons to each other, their removal does not impair the function of the foot

4. Suture the skin margins from side to side and apply a compression bandage Ambulation may begin the next day Healing is usually uneventful.

DISARTICULATION AT FIRST METATARSOPHALANGEAL JOINT

Unlike the advice regarding the lesser toes, the base of the proximal phalanx of the hallux should be saved whenever possible, because of the important intrinsic muscles inserted into it This is especially true of the flexor hallucis brevis, because when the flexor hallucis brevis loses its insertion, it retracts the sesamoids proximally to the middle of the shaft of the first metatarsal.

1 Make a dorsal semilunar incision, starting just behind the head of the first metatarsal

2 Make a second plantar horseshoe incision with its uppermost crest distally, beginning in the first toe web and ending on the medial side of the base of the proximal phalanx This forms a plantar flap which should be made as wide and as long as possible to permit covering the stump without tension

3 (a) If the base of the proximal phalanx can be saved, the bone should be sawed at its proximal epiphyseal line and the surface smoothed and rounded with a rasp Cover the stump with the plantar flap and suture it into place

(b) In the event that the entire proximal phalanx must be removed, after the horseshoe incision has been made, sever the extensor and flexor hallucis longus tendons just proximal to their insertion into the distal phalanx and hold them aside Section the dorsal metatarsophalangeal capsule transversely Plantar-flex the toe Denude the tendons of the adductor, abductor, and flexor hallucis brevis from the base of the proximal phalanx Remove the entire toe

4 Suture the flexor hallucis brevis tendon to the long flexor tendon, suture the long flexor tendon to the long extensor tendon That forms a cover on the articular surface of the first metatarsal head The abductor and adductor hallucis tendons can usually be sutured to each other or to the long tendons

5 Bring up the plantar skin flap to cover the stump and suture it into place

Weight bearing should not be permitted for about three weeks Because the great toe has an important balancing function in walking or running and its loss can affect weight bearing seriously, a shoe with a rigid sole or a rigid inlay, extending the full length of the shoe, should be prescribed Such a shoe helps in balancing the foot.

AMPUTATION OF A METATARSAL

A single metatarsal rarely needs to be amputated. Gangrene or extensive traumatic mutilation usually involves all or multiple metatarsals It is possible, however, for a single metatarsal to be diseased or to be destroyed by injury Removal is then indicated

1 Begin an incision at the base of the metatarsal to be removed and carry the incision distally to the web space, thence through the web, and loop it around the flexor crease of the toe to be removed

2 Continue the course of the incision, through the alternate web, and extend the incision proximally to the starting point of the incision

3 Retract the skin margins and carry the incision down into the intermetatarsal space on both sides of the metatarsal, dividing all soft tissue and the bone

4 Sever the tendons over and under the proximal third of the metatarsal

5 Disarticulate at its base Control bleeding

6 Close the resulting dead space as much as possible

7 Approximate the skin margins side to side and suture

TRANSMETATARSAL AMPUTATION

Transmetatarsal amputation is indicated when there is gangrene of all or a part of one or more toes as a result of vascular disease The operation is indicated, provided the gangrene and any accompanying infection have been stabilized and have not invaded the dorsal or plantar surfaces of the foot The operation may also be performed in cases of crushing injuries of the toes and metatarsal heads, with or without a stabilized open infection in the toes, provided the entire lesion can be excised and primary closure can be obtained Success of the operation depends on circulatory and nutritional adequacy

Before World War II, it was the practice to amputate for a gangrenous toe Either the gangrenous toe was amputated or below-the-knee amputation was done, although there were isolated advocates of transmetatarsal amputation Since then, conservative amputation through the metatarsals in selected cases has reduced morbidity and mortality Pedersen and Day (1954) have reported twenty-three cases of transmetatarsal amputations because of peripheral vascular disease Only two patients had gross failure, requiring subsequent above-the-knee amputation Wheelock and his associates (1957) reviewed sixty-seven of their cases of transmetatarsal amputation for diabetic gangrene and reported that they had only five failures requiring higher amputation at a later date

The preparatory period most importantly determines surgical success For two or three weeks preoperatively the patient is restricted to his bed. During that period, infection must be controlled When infection has been completely controlled and the gangrenous process has been halted, transmetatarsal amputation may be contemplated

The amputation level is just proximal to the metatarsal heads, so that when amputation has been performed, bone remains only between the dorsal and plantar layers of skin and subcutaneous tissue If the level is any more proximal,

it will pass through the deep structures of the foot, including relatively ischemic muscle, which risks further infection and necrosis

1. Begin the dorsal incision midway between the dorsal and plantar surfaces on either side and continue the incision, sharply down to the bone, at the level of amputation, in a straight line across the dorsum to the midpoint on the opposite side (Fig 403, *A*).

2 Begin the plantar incision at either end of the dorsal incision Incise sharply and directly down to the bone. Parallel the proximal flexion crease of the toes, 1 cm proximally to the flexion crease

3 Dissect the plantar flap back to the level of bone amputation

4 With a saw, remove the metatarsal heads, beginning with the first, to form an even stump

5 Remove the plantar tendons and sesamoids at the bone level

6 Closure is preferable in one layer with a nonabsorbable suture Surgical procedure must be as atraumatic and as cautious as possible Skin margins must not be touched with forceps, dog ears, which slough out, must not be left Handle tissues gently with the fingers and a moist gauze sponge

7 Apply a bulky dressing to the whole foot with only slight pressure.

The patient is returned to bed rest Sutures remain in place for two weeks. Buerger's exercises are begun ten days to two weeks after operation Ambulation is begun in three weeks.

REFERENCES

Pedersen, H E, and Day, A J Transmetatarsal Amputation in Peripheral Vascular Disease, J Bone & Joint Surg 36A 1190-1199, Dec, 1954

Wheelock, F C, Jr, McKittrick, J B, and Root, H F Evaluation of Transmetatarsal Amputation in Patients With Diabetes Mellitus, Surgery 41 184-189, Feb, 1957

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